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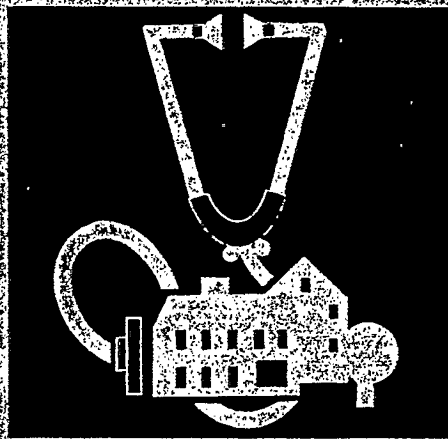

INDOOR AIR POLLUTION

A COMPLETE UPDATE ON IAQ POLLUTANTS, SOURCES EFFECTS, LIABILITIES, RESEARCH, AND CONTROL

MAY 2 - 3, 1991
TULSA, OKLAHOMA

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M.A. Logue

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Division of Continuing Education
The Center for Environmental Research and Technology

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A Complete Update on IAQ
POLLUTANTS, SOURCES, EFFECTS, LIABILITIES, RESEARCH, and CONTROL

May 2-3, 1991 * Tulsa, Oklahoma

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Division of Continuing Education and
Center for Environmental Research and Technology

Fourth National Seminar

on

INDOOR AIR POLLUTION

UPDATE ON THE SOURCES, EFFECTS,
LIABILITIES, RESEARCH, AND CONTROL

MAY 2-3, 1991
Tulsa, Oklahoma

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INDOOR AIR POLLUTION SEMINAR

Speaker Name ______________________
Topic ______________________
Date ______________________
Notes ______________________

AIR DUCT CLEANING

Notes:

Richard Shaughnessy - Twilight Zone - Indoor Air Zone
EPA ranked ~~EPA~~ Radon & Indoor Air Pollutants as
the no. 1 & 2 health risks in the U.S.

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Compliments of

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Mike Etheredge - editor Indoor Pollution News -

INDOOR AIR POLLUTION SEMINAR

Speaker Name Leon Horowitz, M.D.
Topic Indoor Air Quality & Health
Date May 2 8:45
Notes Specializes in Allergy

BREATHE-EASY AIR DUCT CLEANING

Notes:

98% of time indoors, 68% at home - adults
discussed sources of IA problems - Major contaminent
of IAQ is cigarette smoke - more formaldehyde
* in ETS than carpets - the toxic combustion products
* of ETS are greater than main stream - development
of lung is impaired by ETS exposure in children
Radon - most homes low level - EPA 4 pci/lu -
where did EPA get it - it pulled it out of the
air - no one knows what a safe level -
extrapolation from miners exposures.
The important issue - if there is bacteria - amalgam
in the mouth (amalgam in sheep show up in fetus) -
are these contaminents - health hazards?
* Cigarette smokers have a higher rate of nitetime accidents
because CO impairs vision

Name
Address
Phone #

Radon - much money to be made - are the home level significant -
much res. is still needed.

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INDOOR AIR POLLUTION SEMINAR

Speaker Name Mr Robert Axelrad
Topic Federal Indoor Air Program
Date 9/15 5/2
Notes

BREATHE-EASY
AIR DUCT CLEANING

Notes:

90% of time indoors - economic impacts tens of billions
"Pollutant of the year award" as a result of medical attention
Because of so many sources - difficult to identify
Need to understand the health effects of low level exp -
the science isn't there yet
source management is the most effective way to solve problems
OSHA - major surge in issues - ETS & EPA
NIOSH working with EPA on information bulletin
EPA will not ~~reg~~ regulate state efforts - will serve as a resource
Budget - skyrocketed
Next year - each regional will have an IAQ contact
feels more money is needed to characterize health effects

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SARA Acts has allowed EPA to go forth on its ETS issues

MCS - Controversial - EPA will stand on middle of fence
NAS/NRC - March 91 workshop - a report due soon - will be used to obtain funding

Policy Development -
Promote interdisciplinary forum - curriculum - workshops
- at some pt there will be oversight of firms

Source reduction - best

Mitchell/Chafee -
Axelrad feels these bills will not advance the IAQ problems because the science will ~~not~~ take a long time -

Axelrad feels regulation is not the way to go - he wants to develop guidance of

EPA is going forward with guidence to different groups
See A.9

Pollutant / Source Program -
ETS -

2025525564

/OR AIR POLLUTION SEMINAR

Abelrad

Date

Notes

Notes:

ETS - Major Indoor Pollutant -

Exemption for TOXIC

Resp risk - cancer risk

Now - formal lung cancer RA

Life - training courses

self paced training - learning module / ref. manual

intra agency. current fed activities of res.

Risk Characterization is years away - must take immed

mes. to control sources

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INDOOR AIR POLLUTION SEMINAR

Speaker Name John Spengler
Topic
Date 4/2
Notes

BREATHE-EASY
AIR DUCT CLEANING

Notes:

Some people living in "clean air" have a high burden of pollutant exposure due to IAQ
due to the demographics - aging of the population
concern for CN disorders - immune deficiencies -
cardiovascular - health issues for women - for minorities
healthyness in lifestyle will translate to healthyness in the workplace - people will accept voluntary risks much more readily than involuntary risks -
"they will be defined by the courts if govt and employers do not address these risks"

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INDOOR AIR POLLUTION SEMINAR

Speaker Name Spengler
Topic
Date
Notes

BREATHE EASY AIR DUCT CLEANING

Notes:
Comprehensive Indoor Air Policy
1 define Indoor Environments
- Public vs Private
- State public interest & principle for govt intervention
2. Set Reasonable Goals for Govt Action
3 Determine Effective Mechanisms
- Pollution prevention
- exposure reduction
4 Establish IAQ Standards / Guidelines
- category of building
- targeted population

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INDOOR AIR POLLUTION SEMINAR

Speaker Name Spengler -
Topic
Date
Notes

BREATHE EASY AIR DUCT CLEANING

Notes:

Issue of setting standards is very confusing
EPA list Contam in IA -
need to devote much res. to IAQ because
of the time we spend indoors -
homes 65%, Work or school - 15%
discussed home ventilation systems } all decreased
comm build designs } fresh air exchanges
newer airplanes } due to economic cond
"the reduction in air exchanges in airplanes resulted
in the ban of smoking" - Spengler -
health - points of entry of pollutants. inhalation
and health effects general - very basic explanation
of what happens when our bodies are exposed
to environmental health hazards - "insults"
Spengler is tracking
children -

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* Sam t & Spengler - following new homes for 18 mos - measuring lung function every two weeks

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Source of 'HQ' -
Jue -

INDOOR AIR POLLUTION SEMINAR

Speaker Name ______________________
Topic ______________________
Date ______________________
Notes ______________________

BREATHE-EASY
AIR DUCT CLEANING

Notes:

to save time - Spengler skipped over ETS slides
when onto household products -
EPA - personal monitors - many of the
pollutants coming from home exposure:
230 materials ~~can~~ outgas benzene.
730 " " xylene

will lead to product testing & labeling -
Should we be spending money cleaning dump
sites vs addressing IAQ problems

* 45-60% of households in their surveys have severe
mold problems - when childrens' resp. effects were correlated
with those households - high correlation

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* NIH - large new studies on asthma - a great increase
over the last 10 yrs

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* Kirk, Smith Study - 1000 times more BAP than any
smoke-filled bar - new study

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INDOOR AIR POLLUTION SEMINAR

Speaker Name Kevin Teichman
Topic US Fed EPA IAQ Res Prog
Date
Notes Offices inside EPA

BREATHE EASY AIR DUCT CLEANING

Notes:

areas of research:
gas filtration, radon mitigation
TEAM Field Studies
Devils Lake, N.D. vs Bayon, NJ - indoor levels in both higher than outdoor in NJ
- Computer based study
- .92 $-4 ~~billion~~ million for baseline buildings - and development of protocols
* sensory irritants of VOC mixtures
* Eval of Cot as a biomarker of ETS - demonstrate that cotinine can be used to measure ETS exposure
NIOSH - Federal Squat team - 3 year waiting list

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INDOOR AIR POLLUTION SEMINAR

Speaker Name ______________________
Topic ______________________
Date ______________________
Notes ______________________

BREATHE-EASY AIR DUCT CLEANING

Notes:

Office build in DC - 9000 questionaire - available upon request
Teichman -
Uncertainties c Radon - because of extrapolation
DOE is combining results from 16 diff groups world wide
An ounce of prev. is worth 1 lb of cure -
Cause is pol source - not the ventilation -
provide a ventilation for the source that is necessary -

Cfm cubic feet/min/person -

*dramatic increases in vent do not result in dramatic
increase in energy cost - in fact reduced worker productivity
costs go up

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Jim Woods & he wrote a paper in 1986 - on energy & cost consideration

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INDOOR AIR POLLUTION SEMINAR

Speaker Name Barry Reed
Topic Legal Implications of IAQ
Date
Notes

BREATHE-EASY AIR DUCT CLEANING

Notes:

Reilly told Congress that what the public perceives as health hazards & the reality are divergent -

Sources -

- Naturally Occuring - radon
- building materials
- activity related (tobacco smoke/occupational)

- sick building syndrome

synergistic effects

Regulation of IAQ

- Government - relatively little -

govn works most effectively with crisis not yet

- Government has focused on public inf res, remediation

Reason - there may not be a need for govt to regulate - the market place (i.e. legal) may take care of it

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INDOOR AIR POLLUTION SEMINAR

Speaker Name Lawyer - Texas
Topic
Date
Notes

BREATHE-EASY AIR DUCT CLEANING

Notes:

Radon Program - EPA
- 4pci/L guidance for remedial action
- standardization of methods

- Toxic Substance Control Act (TSCA) for manufactured products
State regulations of radon
research, education, testing, const standards
Asbestos - change in EPA recommendation - management vs removal

ASHRE Standards are not legally enforceable - but courts will use the acceptable standards

element of liability
- abnormally dangerous product & product defect

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INDOOR AIR POLLUTION SEMINAR

Speaker Name ______________________
Topic ______________________
Date ______________________
Notes ______________________

BREATHE-EASY
AIR DUCT CLEANING

Notes:

lawyers recommendation -
- awareness including scientific data
must remain current on

-

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INDOOR AIR POLLUTION SEMINAR

Speaker Name Alan Hedge -
Topic
Date
Notes

BREATHE-EASY AIR DUCT CLEANING

Notes:

symptoms of SBS are all conjecture
SBS - Building Risk Factors from EPI studies (Increase symptoms)
- HVAC System
- Open-plan offices
- Office Lighting (fluorescent)
- Evaporative or spray humidifiers

SBS - Unrelated factors
- ventilation rates
- carbon monoxide & dioxide levels
- VOC & formaldehyde levels
- negative air ion levels

all these contaminants who targeted these or Alan

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INDOOR AIR POLLUTION SEMINAR

Speaker Name ____________________
Topic ____________________
Date ____________________
Notes ____________________

BREATHE-EASY
AIR DUCT CLEANING

Notes:

? S.S. Envion Risk Factors
- air temp above 22°C
- humidity > 50%
- fleece & shelf factors
- ETS exposure - no relation -
his recent studies of sick buildings
are smoke free

* coffee-drinkers complain the most
govt building report more symptoms
clerical workers
stressful jobs } complain more
competitors opp } more symptoms

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INDOOR AIR POLLUTION SEMINAR

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Topic ______________________
Date ______________________
Notes ______________________

BREATHE-EASY
AIR DUCT CLEANING

Notes:

Alan Hedge - good presentor - not particularly believable?
While made last year -

-

sensation → discrimination - decision

* currently doing a survey on 18 buildings

Changes in psychological impact changes
in biochemistry

Alan Hedge said some very controversial things
Breathing zone system - installed in NJ
localized filtration

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INDOOR AIR POLLUTION SEMINAR

Speaker Name Hal Levine
Topic
Date
Notes

BREATHE-EASY AIR DUCT CLEANING

Notes:

Indoor Air '91 attended by 100 people -
Environmental Engineers, Building engineers and
building mitigators
Reports to complete
Radon Meeting
Dod - Risk Assessment
Science and Ethics/Research
It is clear that many of the questions from
the audience concerned with specific illness
associated with particular pollutants
Hal Levine talked about design of building -
one must design a body building to decrease
stress -

Stress decreasing
ability to deal with
pollutants, noise &
light

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INDOOR AIR POLLUTION SEMINAR

Speaker Name ______________________
Topic ______________________
Date ______________________
Notes ______________________

Notes:

Centria ~~fo~~
air brought in at workers desk -
can be individually controlled

Electrostatic filter used in bars, etc
to remove 0.6 - 1.2 u particles

Source control is more effective
than ventilation (~~source cont~~ dilution)
Levin talked about encapsulating fiberboard -
using paint which has lower emissions

Manufacturers will have to disclose emissions
before purchase of
carpets / ~~workshops~~
e workstations

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INDOOR AIR POLLUTION SEMINAR

Speaker Name Harriet Burg
Topic Bioaerosols
Date 5/3/91
Notes

BREATHE-EASY
AIR DUCT CLEANING

Notes:

Alfatoxin - Carcinogenic form produced by fungi
dust mites - most probable cause of childhood asthma

with SBS - you must consider psycogenic illness
Building managers can go a long way to diffuse the situation especially c̄ psycogenic

House dust mites
env. control - low relative humidity
- limit carpeting

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INDOOR AIR POLLUTION SEMINAR

Speaker Name
Topic
Date
Notes

BREATHE EASY
AIR DUCT CLEANING

Notes:

Centria - fp
air brought in at workers desk -
can be individually controlled

Electrostatic filter used in bars, etc
to remove 0.6 - 1.2 u particles

Source control is more effective
than ventilation (~~source cont~~ dilution)
Levin talked about encapsulating fiberboard -
using paint which has lower emissions

Manufacturers will have to disclose emissions
before purchase of
carpets / ~~workshops~~
& workstations

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INDOOR AIR POLLUTION SEMINAR

Speaker Name ______________________

Topic ______________________

Date ______________________

Notes ______________________

BREATHE-EASY
AIR DUCT CLEANING

Notes:

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THE UNIVERSITY OF TULSA
Division of Continuing Education

WELCOME!

The University of Tulsa's Division of Continuing Education and The Center for Environmental Research and Technology, welcomes you to the fourth national seminar on **"INDOOR AIR POLLUTION: A Complete Update on IAQ POLLUTANTS, SOURCES, EFFECTS, LIABILITIES, RESEARCH, AND CONTROL"**, May 2-3, 1991.

The program will be held from 8:00 a.m. to 5:30 p.m. each day. There will be daily refreshment breaks which will give you an opportunity to visit informally with the instructors, the other participants, and the corporate sponsors. Be sure to take time to visit the sponsors' booths.

This is your program and your participation is encouraged. Please ask any questions that you feel are relevant and helpful to the understanding of the material being presented.

Please take a few minutes at the end of the seminar to fill out the evaluation form provided. The University of Tulsa values your opinion and suggestions for the purpose of future offerings on this topic.

THANK YOU for your attendance. We appreciate your interest in our Continuing Education programs. We want this seminar to be an educational and enjoyable experience. Please do not hesitate to call on us if you have any questions.

Sincerely,

Richard Shaughnessy, Program Chairman
Pat Hall, Assistant Dean & Director, Continuing Engineering Education
Nancy Felts, Program Coordinator, Continuing Engineering Education
Kerry Sublette, Center for Environmental Research and Technology

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The University of Tulsa's Division of Continuing Education and The Center for Environmental Research and Technology gratefully express our appreciation to the

CENTER FOR INDOOR AIR RESEARCH

for its support of our conference this year.

CENTER FOR INDOOR AIR RESEARCH

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PROGRAM SCHEDULE

Thursday, May 2, 1991

8:00 - 8:30 a.m.	**Richard Shaughnessy, Ph.D.** Introduction
8:30 - 9:00 a.m.	**Leon Horowitz, M.D.** Indoor Air Quality and Health
9:00 - 9:45 a.m.	**Robert Axelrad** EPA's Program on IAQ
9:45 - 10:00 a.m.	Break
10:00 - 11:15 a.m.	**John Spengler, Ph.D.** Overview on Indoor Air Quality
11:15 - 12:15 p.m.	**Terry Brennan** Radon Control and IAQ Concerns in Underventilated Buildings: School Studies
12:15 - 1:00 p.m.	Lunch on your own
1:00 - 2:00 p.m.	**Kevin Teichman, Ph.D.** The U.S. Federal EPA IAQ Research Program
2:00 - 3:00 p.m.	**Thad Godish, Ph.D.** Residential IAQ Problems
3:00 - 3:15 p.m.	Break
3:15 - 4:15 p.m.	**Laurence Kirsch, Esq.** Current Legal Trends on Indoor Air
4:15 - 5:00 p.m.	**Alan Hedge, Ph.D.** Psychosocial and Environmental Influences on Sick Building Syndrome
5:00 - 5:45 p.m.	**Panel Discussion**

Friday, May 3, 1991

8:00 - 9:30 a.m.	**Harriet Burge, Ph.D.** Bioaerosols
9:30 - 10:45 a.m.	**Philip Morey, Ph.D., C.I.H.** Building Performance Relative to Bioaerosols and VOCs
10:45 - 11:00 a.m.	Break
11:00 - 12:15 p.m.	**Charlene Bayer, Ph.D.** Operation and Maintenance and Its Effects on Healthy Buildings
12:15 - 1:00 p.m.	Lunch on your own
1:00 - 2:30 p.m.	**William A. Turner** Diagnostic & Investigative Techniques
2:30 - 2:45 p.m.	Break
2:45 - 4:30 p.m.	**Hal Levin** Prevention and Control of Indoor Air Quality Problems
4:30 - 5:30 p.m.	**Panel Discussion**

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ACCREDITATION INFORMATION

COURSE: INDOOR AIR POLLUTION: A complete update on IAQ POLLUTANTS, SOURCES, EFFECTS, LIABILITIES, RESEARCH, AND CONTROL

DATE: May 2-3, 1991

LOCATION: Tulsa, Oklahoma

INSTRUCTORS:

Robert Axelrad
Terry Brennan
Thad Godish, Ph.D.
Leon Horowitz, M.D.
Hal Levin
John Spengler, Ph.D.
William Turner
Charlene Bayer, Ph.D.
Harriet Burge, Ph.D.
Alan Hedge, Ph.D.
Laurence Kirsch, Esq.
Philip Morey, Ph.D., C.I.H.
Kevin Teichman, Ph.D.

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The Center for Environmental Research and Technology

INTRODUCTION

Speaker:

RICHARD SHAUGHNESSY, Ph.D.
Program Chairman

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THE UNIVERSITY OF TULSA
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Dr. Richard Shaughnessy received his Ph.D. in Chemical Engineering at The University of Tulsa. He is Program Manager for indoor air research under the Center for Environmental Research & Technology (C.E.R.T.) at the university and has long served as spokesman for the university on indoor air and radon related subjects.

Dr. Shaughnessy served as advisor to the Governor of Oklahoma's Environmental Concerns Council in 1988 and was instrumental in providing background information and recommendations on indoor air quality to the Governor. He has been the principal coordinator for four national seminars on Indoor Air Pollution from 1988 - 1991, and has served as a consultant on IAQ investigations throughout the region in the past five years.

His present activities on IAQ under C.E.R.T. include the following educational and research efforts:

EDUCATIONAL EFFORTS

-General public outreach and educational efforts on indoor air quality and radon.
-Development of realtors' course on indoor air quality, radon, and associated liability.
-Coordination of annual, national two-day seminars on indoor air quality.
-Assisting EPA in implementation of presenting two-day courses "Orientation to IAQ".
-Training with respect to radon, as a consortium member of EPA's Southern Regional Radon Training Center.
-Establishing IAQ network of professionals (directed by EPA's Region VI office) within Region VI to enhance outreach and assess IAQ needs within the region.

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RESEARCH EFFORTS

- IAQ related to hospitals, clean rooms, and automobiles.
- Evaluation of effectiveness of portable air cleaners on removal of common IAQ contaminants.
- EPA funded innovative radon project.
- Development of IAQ research strategy to include buildings inventory and associated demographics study within the region.
- Development of database of IAQ within schools in the U.S.

As Program Chairman, Moderator, and Speaker at the national IAQ seminars held in Tulsa, Dr. Shaughnessy has developed close ties with local and state governments, business associations, building associations, environmental associations, the American Lung Association, and other interested groups in IAQ across the country. He hopes to strengthen and expand upon these ties in the future to enable the university to further develop its educational and research efforts on indoor air.

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Sources of Further Information

Books

Ashford, Nicholas, Ph.D., J.D., and Claudia Miller, M.D., Chemical Exposures: Low Levels and High Stakes. New York: Van Nostrand Reinhold, 1991.

Bower, John, The Healthy House. New York: Carol Communications, 1989.

Cone, James E., M.D., M.P.H., and Michael J. Hodgon, M.D., M.P.H., eds. Problem Buildings: Building-Associated Illness and Sick Building Syndrome. Hanley and Belfins, Inc., 210 South 13th Street, Philadelphia, PA 19107 1989 215-546-7293.

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Hayes, Wayland J., M.D., PhD., Pesticides Studied in Man. Baltimore: Williams and Wilkins, 1982.

Health and Safety in Buildings through Technology. NIBS Publications Department, 1201 L Street, N.W., Suite 400, Washington, D.C. 20005 202-289-7800.

Lawrence Berkeley Laboratory, Manual for Indoor Air Quality. Research Reports Center (RRC), Box 50490, Palo Alto, CA 94303 412-965-4081.

Nagda, N. L., and J. P. Harper, Design and Protocol for Monitoring Indoor Air Quality. ASTM, 1916 Race Street, Philadelphia, PA 19103 1989.

National Research Council, Indoor Pollutants. National Academy Press, 2101 Constitution Ave. N.W., Washington, D.C. 20418 1981.

Nero, Anthony, et al.. Indoor Radon and Decay Products: Concentrations, Causes, and Control Strategies. U.S. Department of Energy, 1990

Standards, Regulations, and Other Technical Criteria Related to Indoor Air Quality. NIBS Publications Department. See above.

Turiel, Isaac, Indoor Air Quality and Human Health. Stanford, Ca: Stanford University Press, 1983.

Wadden, Richard A., and Peter A Scheff, Indoor Air Pollution. New York: John Wiley and Sons, 1983.

Wallace, Lance, The Total Exposure Assessment Methodology (TEAM) Study: Summary of Analysis. Vol. I. Office of Research and Development, USEPA, Washington, D.C. 20460 1987.

Walsh, Phillip J., ed. et al., Indoor Air Quality. CRC Press. See above.

Workers with Multiple Chemical Sensitivities. Hanley and Belfins, Inc. 1987. See above.

Sources of Further Information

Other Publications

ASHRAE, "Ventilation for Acceptable Indoor Quality: ASHRAE Standard 62." ASHRAE, 1791 Tullie Circle, N.E., Atlanta, GA 30329 404-636-8400.
EPA, "Current Federal Indoor Air Quality Activities."
EPA, "Indoor Air Fact Sheets 1-7."
EPA, "Information on Levels of Environmental Noise Requisite to Protect Public Health and Welfare with an Adequate Margin of Safety." 1974.
EPA, "The Inside Story."
EPA, "Residential Air-Cleaning Devices: a Summary of Available Information."
NIOSH, U.S. Department of Health and Human Services, "Indoor Air Quality, Selected References, 1989, Testimony of J. Donald Millar, M.D., Director of NIOSH, before the Subcommittee on Superfund, Ocean and Water Protection Committee on Environment and Public Works, U.S. Senate, May 26, 1989."

Periodicals

The Indoor Air Quality Update, 37 Broadway, Arlington, MA 02174-5539 617-648-8700.
Indoor Pollution Law Report, Leader Publishing Company, Inc., Box 29, West 2nd Street, Coudersport, PA 16915 814-272-8044.
The Indoor Pollution News, Buraff Publications, 1350 Connecticut Ave, N.W., Suite 1000, Washington, D.C. 20036 202-862-0990

Topics to use to find more information in a library or database

indoor air quality
indoor air pollution
pollution, indoor
pollution, air, indoor
environmental health
environmental disease
environmental illness
environmentally-induced disease
housing and health
building-related illness
sick building syndrome
sick building
chemical sensitivity
multiple chemical sensitivity (MCS)
You can also look up the name of a specific compound.

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Sources of Further Information

Organizations

American Cancer Society
American Heart Association
American Lung Association
City and county health departments
ASHRAE, local and national chapters
Consumer Product Safety Commission, Product Safety Hotline, 1-800-638-CPSC
EPA Regional Office, Region 6, Carrie Paige, 214-655-7214
Human Ecology Action League, P.O. Box 49126, Atlanta, GA 30359-1126
National Institute for Occupational Safety and Health
National Pesticides Telecommunications Network, 1-800-858-PEST
Occupational Safety and Health Admininstration
US EPA INDOOR AIR DIVISION, 1--703-3088470

UNIVERSITY OF TULSA, CENTER FOR ENVIRONMENTAL RESEARCH
& TECHNOLOGY, DR. RICHARD SHAUGHNESSY, 1-918-7494358
600 S. COLLEGE AVE.
DEPT. OF CHEMICAL ENG.
TULSA,OK. & 74104-3189

The Chemical Connection, RR1 Box 276A65, Wimberley, TX 78676 512-847-9245
World Health Organization
Additional organizations are listed in "The Inside Story," pp. 29-31.

Indoor Air Quality
Document Mailing List

Please check the document(s) you would like to receive:

- ______ The Inside Story
- ______ Residential Air-Cleaning Devices
- ______ Indoor Air Facts #1: EPA and Indoor Air Quality
- ______ IAF #3: Ventilation and Air Quality in Offices
- ______ IAF #4: Sick Buildings
- ______ IAF #5: Environmental Tobacco Smoke
- ______ IAF #6: Report to Congress on Indoor Air Quality
- ______ IAF #7: Residential Air Cleaners

Name: ______________________________

Affiliation: ______________________________

Address: ______________________________

City, State, Zip: ______________________________

Telephone: ______________________________

SEND REQUEST TO:

Indoor Air Quality Program
US EPA, Region 6 Office
Mail Code 6T-ET
1445 Ross Avenue
Dallas, TX 75202

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THE UNIVERSITY OF TULSA
Division of Continuing Education
The Center for Environmental Research and Technology

Indoor Air Quality and Health

Speaker:

LEON HOROWITZ, M.D.

Allergy Clinic of Tulsa
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LEON HOROWITZ, M.D.

Leon Horowitz, M.D. is a native New Yorker who has lived in Tulsa since 1955. Dr. Horowitz received his medical degree from New York University and his pediatric training at Lenox Hill and Bellevue Hospitals in New York City.

His allergy training was at the Kaiser Permanente Hospital in San Francisco. Dr. Horowitz practiced general pediatrics in Tulsa from 1955 through 1958 and pediatric allergy from 1959 to the present. Dr. Horowitz is Certified by the American Board of Pediatrics and the Sub-Board in Pediatric Allergy and by American Board of Allergy & Immunology. He is a Fellow of the American Academy of Pediatrics, the American Academy of Allergy & Immunology and the American College of Allergists. He is recipient of the AMA Physicians Recognition Award. Dr. Horowitz is Clinical Professor of pediatrics at the University of Oklahoma Tulsa Medical College and Director of its Pediatric Allergy & Immunology Clinic at the Tulsa Ambulatory Pediatric Center.

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THE UNIVERSITY OF TULSA
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The Center for Environmental Research and Technology

Environmental Protection Agency's Program on Indoor Air Quality

Speaker:

ROBERT AXELRAD

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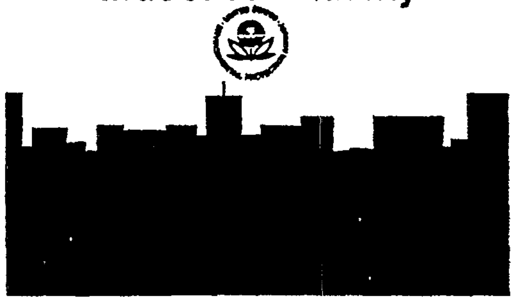
Indoor Air Quality



Indoor Air Quality

- The Problem
- The Solutions
- The Program

Indoor Air Quality: The Problem

Why is Indoor Air Important?

- Indoor levels often higher than outdoors
  - 2-5 times higher for many pollutants
  - Up to 1000 times higher during and shortly after use
- People spend app. 90% of time indoors
- Significant short & long term health impacts
- Economic impacts in tens of billions of dollars per year

Indoor Air Quality

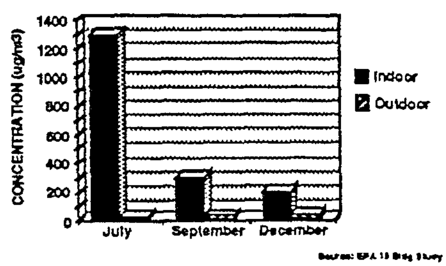
Long Term Risks.

- Asbestos
- Radon
- ETS

Immediate Effects

- Building Related Illness
- Sick Building Syndrome

Total Organics in a New Office Building



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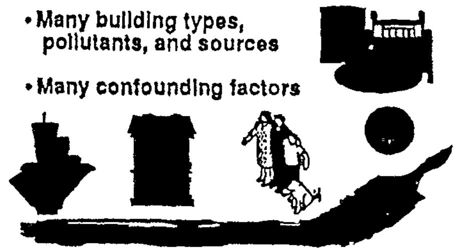
Comparing Risks to Set Environmental Priorities

- 1987 Comparative Risk Project: Indoor Air Ranked #4
- 1989 Regional Rankings Indoor Air Ranked #2
- 1990 Science Advisory Board Indoor Air Among Top 4

√Indoor Air Quality is a Complex Problem

- Many building types, pollutants, and sources
- Many confounding factors



Pollutants and Pollutant Sources

Pollutant	Source(s)
Radon and radon progeny	Soil, well water some building materials
Environmental tobacco smoke (ETS)	Tobacco smoking
Biological contaminants (bacteria, viruses, fungi, mold spores, pollen, other biological material)	Outdoors, humans, animals, HVAC systems
Volatile organic compounds (VOCs)	Paints, stains, adhesives, dyes, solvents, caulks, cleaners, pesticides, building materials

√ Pollutants and Pollutant Sources

Pollutant	Source(s)
Formaldehyde	ETS, UFFI, particle board, plywood, furnishings, upholstery
Polycyclic aromatic hydrocarbons (PAHs)	ETS, kerosene heaters
Pesticides	Pesticide application indoors and outdoors
Asbestos	Asbestos cement, insulation, other building materials

Pollutants and Pollutant Sources

Pollutant	Source(s)
Combustion Gases	
• Carbon Monoxide (CO)	Combustion appliances, ETS, infiltrated exhaust
• Nitrogen Dioxide (NO_2)	Combustion appliances, ETS
• Sulfur Dioxide (SO_2)	Combustion of fuels containing sulfur (e.g. kerosene heaters)
Particles	Combustion appliances, ETS, personal activities (e.g. sprays, cooking aerosols)

√Factors Affecting Occupant Perception of Indoor Air Quality

- Chemical contaminants
- Biological contaminants
- Ventilation (outside air supply & air movement)
- Thermal comfort (temp / humidity)
- Odor
- Psycho-social factors
- Lighting / ergonomics / other factors

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Additional Complexities

- Limited scientific understanding of many health effects issues:
  - Mixtures
  - Low levels of pollutants
- Ventilation assessments complex

Indoor Air Quality:
The Solutions

Indoor Air Quality Solutions

Hierarchy of IAQ Control Strategies

- Source Management
  - Lower emitting materials
  - Removal, encapsulation, time of use, etc.
- Ventilation
  - Dilution
  - Exhaust
  - Comfort
- Air Cleaning

Indoor Air QualitySolutions

Prevention

- Designate a responsible person
- Develop an IAQ profile:
  - Existing records
  - Walkthrough inspection
- Develop an IAQ management plan:
  - Assign staff responsibilities
  - Maintenance & operations
  - Occupant activities

Indoor Air Quality Solutions

Problem Solving

- Collect information on:
  - Occupant complaints
  - HVAC system
  - Pollutant sources & pathways
- Form & test hypotheses
- Design & implement mitigation strategies

Indoor Air Quality:
The Program

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√ Federal Agencies Involved in Indoor Air Quality

Environmental Protection Agency (EPA)
Consumer Product Safety Commission (CPSC)
Department of Energy (DOE)
National Institute for Occupational Safety & Health (NIOSH)
Occupational Safety & Health Administration (OSHA)
General Services Administration (GSA)
Department of Transportation (DOT)
Department of Housing & Urban Development (HUD)
Department of Commerce (DOC)
Department of Defense (DOD)
National Aeronautics & Space Administration (NASA)
Plus approximately 8 other agencies

√ Federal Agencies Involved in Indoor Air Quality

Environmental Protection Agency (EPA)

- Broad research and information authority
- Statutes to control individual chemicals / pesticides
- Major programs to address asbestos, radon

√ Federal Agencies Involved in Indoor Air Quality

Consumer Product Safety Commission (Independent Agency)

- Consumer product regulations

Occupational Safety and Health Administration (Dept. of Labor)

- Worker health & safety regulations
- Primarily industrial workplaces

Federal Agencies Involved in Indoor Air Quality

National Institute of Occupational Safety & Health (Department of Health & Human Services)

- Building investigations
- Research
- Guidance

√ Federal Agencies Involved in Indoor Air Quality

Department of Energy (DOE)

- Energy related research

General Services Administration (GSA)

- Responsibility for Federal Bldgs

Department of Transportation (DOT)

- Airline cabin air quality, other transportation

plus app. 10 other Federal agencies

State Indoor Air Quality Programs

- Decentralized
- Some regulatory programs
- Require technical and information support

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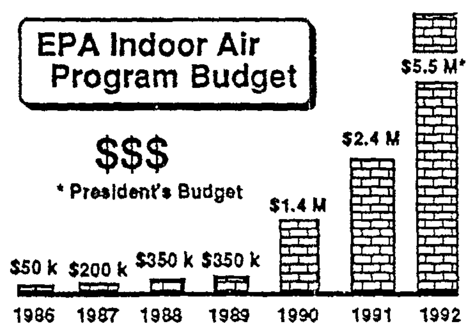
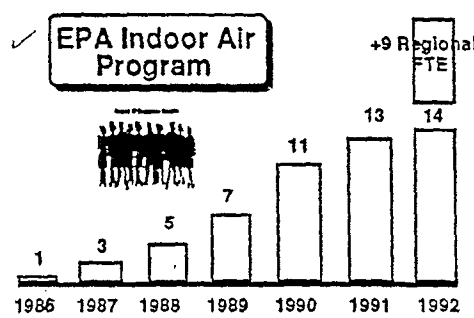
EPA and Indoor Air

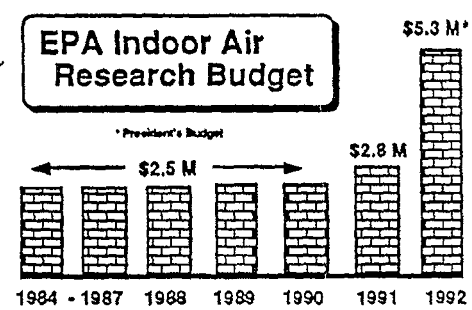
1970	Clean Air Act
70's/80s	Asbestos, formaldehyde
1984	Congressional research $$
1985	Radon Action Program
1986	Sara Title IV: 1st legislation on IAQ
1990	Senate bill passes
1991	New bills introduced

EPA Offices w/ IAQ Exposure Activities

- Office of Air and Radiation (indoor air, radon)
- Office of Research and Development (monitoring, health, and engineering labs)
- Office of Toxic Substances -- to become a major focal point - (asbestos, lead, formaldehyde, other toxic chemicals)
- Office of Pesticide Programs to assess toxic substances
- Office of Drinking Water







Superfund Amendments and Reauthorization Act (SARA) Title IV

Key Elements:

- No regulatory authority
- Emphasis on research and information dissemination
- Limited resource authorizations

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EPA Indoor Air Program

5 Program Elements:
Policy Development
Buildings Program
Pollutant /Source Program
Intergovernmental Program
Public Information Program

EPA Indoor Air Program

5 Program Elements:
Policy Development
Buildings Program
Pollutant /Source Program
Intergovernmental Program
Public Information Program

Policy Development

Goals:

- Identify and fill information gaps for long term policy development
- Establish and implement near term operating principles

Policy Development

Report to Congress on Indoor Air Quality

- Summary of federal activities
- Status of IAQ knowledge
- Research needs
- 6 Recommendations:
  - research
  - guidance, information development/dissemination

Policy Development

Multiple Chemical Sensitivity Research Agenda

- NAS/NRC
- March 91 Workshop
- Identify current knowledge & research needs
- NOT intended to draw conclusions on MCS

Policy Development

Study of Indoor Allergens

- NAS/NRC
- 18 month project
- Identify current knowledge & research needs

Policy Development

Indoor Air Quality Interdisciplinary Forum

- Extension of IAQ firms survey & credentialling project
- Intended to promote cross-fertilization across IAQ disciplines; and
- Enhance existing credentialling, continuing education, and curriculum development programs

Policy Development

FY 92-95 Large Building Study

- Need for baseline data
- Methods development / standardization
- Planning now underway

Initiate study for baseline data (planning) I & me to get data in & out of gov. to develop protocol of NBI? multi-year -

Present Policy Strategy

- Transfer what we know NOW about risk and risk reduction
- Take immediate steps to:

  MINIMIZE HUMAN EXPOSURE TO INDOOR AIR CONTAMINANTS

Present Policy Strategy

- Stress building design, operation, and maintenance as most practical approach to exposure reduction
- Encourage voluntary industry efforts to reduce source emissions
- Improve TSCA/FIFRA pollutant - specific programs

Pending Legislation S. 455 (Mitchell/Chafee)

- No regulatory authority
  - $48.5 million/yr *authorized*
  - Expanded research and research demonstrations
- Health advisories
  - State grants
  - National Response Plan
- NIOSH building investigation program
- Information Clearinghouse

Pending Legislation H.R. 1066 (Kennedy)

- Most provisions of Mitchell Bill
- Also proposes new regulatory programs:
  - Contaminant Emissions Product Labeling - only for prod that contain health risks
  - Ventilation Standards for Public and Commercial Buildings CFm 20 cfm records keeping
  - Workplace Indoor Air Quality Protection OSHA reevaluate - lack of data thus far

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Pending Legislation H.R. 1066 (Kennedy)

Contaminant Emission Product Labeling

- EPA to establish standard test methods for emissions from sources that "pose significant adverse health effects"
- Affected industries to label products with emissions data

Pending Legislation H.R. 1066 (Kennedy)

Contaminant Emission Product Labeling

Problems:

- Presupposes provable adverse health effects of common products
- Attempts to legislate simple fix to TSCA

Pending Legislation H.R. 1066 (Kennedy)

Ventilation Standards for Public and Commercial Buildings

- Mandates minimum of 20 cfm/occupant in all new buildings (design & operation)
- Recordkeeping for all new and existing buildings
- OSHA to enforce
- EPA to evaluate need for pollutant-specific standards

Pending Legislation H.R. 1066 (Kennedy)

Workplace Indoor Air Quality Protection

- OSHA to evaluate need for IAQ standards for the workplace

Pending Legislation

EPA/Administration Position

- Existing authorities sufficient
  - –TSCA performance should be enhanced, *not* replicated
- Resources for IAQ increasing
- Bills would disrupt current effort:
  - –Pollutant specific provisions would produce only controversy, *not* risk reduction
  - –Bills would shift emphasis away from building performance and key audiences and into process, reports and studies

EPA Indoor Air Program

5 Program Elements:

Policy Development

Buildings Program

Pollutant /Source Program

Intergovernmental Program

Public Information Program

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Public & Commercial Buildings

Building Managers' Prevention & Problem- Solving Guidance

- Joint EPA/NIOSH "self-help" guides
- ✓ External review completed
- Completion: Fall 1991

one guide

Public & Commercial Buildings

Architect & Engineers Introduction to IAQ

- Non-prescriptive information
- Entering outside review
- Completion: Early 1992

Schools

Introduction to IAQ for school facility managers

- Concise intro to school IAQ issues
- Completion: Mid 1992

Residential Buildings

Homebuilder's Guide to IAQ

- Basic IAQ design features
- Possible "upgrade" features
- Completion: early 1992

EPA Indoor Air Program

5 Program Elements:

Policy Development

Buildings Program

$\boxed{\text{Pollutant /Source Program}}$

Intergovernmental Program

Public Information Program

Pollutant/Source Program

Environmental Tobacco Smoke

- Health Effects of Passive Smoking: Lung Cancer in Adults and Respiratory Disorders in Children
- Guide to Workplace Smoking Policies
- SAB report April 18th - Rec. changes to poster
- Final documents: Fall 91

Class A carcinogen

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Pollutant/Source Program

Why Environmental Tobacco Smoke?

- Represents significant gap in EPA chemical control program under TSCA
- Cigarettes are a strong source of particles indoors
- Demonstrable acute effects and a strong cancer case
- Not an integral component of building function (e.g. as compared to bldg materials & furnishings)

Pollutant/Source Program

ETS Policymaker's Guide

- Guidance on workplace smoking policies
- Theme: Prudent to minimize exposure
- Basis: 1986 Surgeon General, NRC reports; overall body of literature on ETS:
  - Sensory reactions and irritation effects
  - Lung cancer risk
- Completion: Fall 1991

Pollutant/Source Program

ETS Risk Assessment

(ORD Human Health Assessment Group)

- Purposes:
  1) To evaluate new ETS studies since 1986;
  2) To classify ETS according to EPA carcinogenicity guidelines; and
  3) To assess extent of risk associated with exposure to ETS (lung cancer; respiratory disorders in children)
- Completion: Fall 1991

Pollutant/Source Program

Carpet Policy Dialogue

- Goal to promote low cost/low impact TVOC emission reductions from carpet products:
  - -- carpet
  - -- adhesives
  - -- padding
- Standardized chamber test method

Pollutant/Source Program - Short -

Physician's Guide to Indoor Air Quality

(American Lung Association)

- Intro to IAQ for primary care physicians
- Signs and symptoms, sources, solutions
- Completion date: Early 1992

EPA Indoor Air Program

5 Program Elements:

Policy Development

Buildings Program

Pollutant/ Source Program

Intergovernmental Program

Public Information Program

Intergovernmental Program

Training &Tech Assistance

- "Live "training course for state and local government personnel
- "Self-paced" training module
  - Learning module
  - Reference manual
- Regional program development

be ready this summer

Intergovernmental Program

- Interagency Committee on Indoor Air Quality (CIAQ)
  - *Current Federal Activities*
- NATO/CCMS Pilot Study on Indoor Air

EPA Indoor Air Program

5 Program Elements:

Policy Development

Buildings Program

Pollutant /Source Program

Intergovernmental Program

Public Information Program

Public Information Program

The Inside Story ...
A Guide to Indoor
Air Quality

Public Information Program

Highlights:

- *Directory of State IAQ Contacts*
- Fact Sheets ETS
- Pamphlet on Residential Air-Cleaners
- Information Clearinghouse - database

on line this summer

Summary

- Sufficient evidence exists that indoor air pollution poses significant acute and chronic health risks;
- Complete risk characterization years away;
- Prudent to minimize human exposures through:
  - Building design & operation
  - More effective use of existing chemical-specific program authorities

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THE UNIVERSITY OF TULSA
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The Center for Environmental Research and Technology

Overview on Indoor Air Quality

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1 July 1983, Volume 221, Number 4605 SCIENCE

Indoor Air Pollution: A Public Health Perspective

John D. Spengler and Ken Sexton

Indoor air quality in nonoccupational settings has received relatively little attention from scientists, engineers, regulatory officials, and environmental groups. However, the available information suggests that elevated concentrations of some airborne contaminants are routinely encountered in indoor environments. Time-budget surveys have shown that most of us are at home more than 16 hours per day, with other indoor time divided between the workplace, commercial and public buildings, and transportation microenvironments (for instance, automobiles and subways) (*1*, *2*). Even if indoor air pollutant concentrations are low, they may make a substantial contribution to time-weighted exposures. The degree to which indoor air pollution represents a public health hazard has not been established. Nevertheless, concerns about health effects have prompted intervention at local, state, and federal levels of government to limit indoor exposures to sidestream tobacco smoke, asbestos, formaldehyde, and radon. In this article we discuss the scientific basis for recognizing indoor air pollution as a national health concern and the need for a coordinated policy to safeguard indoor air quality.

Summary. Although official efforts to control air pollution have traditionally focused on outdoor air, it is now apparent that elevated contaminant concentrations are common inside some private and public buildings. Concerns about potential public health problems due to indoor air pollution are based on evidence that urban residents typically spend more than 90 percent of their time indoors, concentrations of some contaminants are higher indoors than outdoors, and for some pollutants personal exposures are not characterized adequately by outdoor measurements. Among the more important indoor contaminants associated with health or irritation effects are passive tobacco smoke, radon decay products, carbon monoxide, nitrogen dioxide, formaldehyde, asbestos fibers, microorganisms, and aeroallergens. Efforts to assess health risks associated with indoor air pollution are limited by insufficient information about the number of people exposed, the pattern and severity of exposures, and the health consequences of exposures. An overall strategy should be developed to investigate indoor exposures, health effects, control options, and public policy alternatives.

Contaminated indoor air is not new. Soot found on ceilings of prehistoric caves provides evidence of the high levels of pollution associated with inadequate ventilation of open fires. Elevated indoor pollutant concentrations continue to be a fact of life for people who live in impoverished areas and cook over open fires fueled by charcoal, wood, dung, kerosene, or oil. Exposures to gaseous and particulate pollutants for these people greatly exceed measured pollutant concentrations in urban environments (*3*).

In industrialized countries, the more obvious indoor pollution problems have been known for decades. Minimum ventilation requirements, for instance, are a standard component of building codes to control odors and combustion by-products and reduce transmission of respirable diseases. However, the complex nature of indoor nonoccupational environments in technologically advanced nations (including synthetic building materials, energy-efficient buildings, unvented heating and cooking appliances, and cleaning and personal care products) makes possible widespread indoor exposures to a broad spectrum of airborne chemicals. Although many indoor pollutants (such as radon decay products and microorganisms) are below perception thresholds, long-term exposures may cause increased rates of morbidity and mortality.

In the 10 years since Benson *et al.* (*4*) reviewed the subject, a number of reports, symposia, and articles (*5–12*) have been published which contain information on indoor contaminants with potentially negative health effects and on important indoor emission sources (see Table 1). Nevertheless, development of public policy to safeguard indoor air quality is hindered by a paucity of data on distributions of sources, building characteristics, daily activity patterns, indoor concentrations, exposure patterns, and health risks.

Basis for Concern

Increases in energy prices and in costs of new electricity-generating stations have encouraged individuals and institutions to seek alternative fuels and to reduce energy consumption. Because more than one-third of U.S. energy is consumed in buildings (*13*), efforts have been made to reduce energy use in the residential and commercial sectors (*14–16*). Common approaches include: adding insulation, reducing air-exchange rates, and fuel switching.

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Increased sales of wood- and coal-burning stoves and kerosene heaters suggest a national trend away from clean space-heating fuels such as electricity and gas. Sales of wood-burning stoves in the United States increased from fewer than 200,000 in 1972 to approximately 1.5 million in 1981 (*17*). An estimated 3 million kerosene heating units were in use during the 1981–1982 winter, with projections of 8 million to 10 million by 1985 (*18*). Emissions from these heating appliances contain toxic and carcinogenic particles and gases (*19–23*), yet indoor exposures and possible health consequences have not been evaluated adequately.

Because hospitals consume approximately 15 percent of all energy used in the commercial building sector, they have been a target for energy savings (*24, 25*). However, a panel of experts who explored the opportunities and constraints associated with relaxation of Department of Health, Education, and Welfare standards concluded that "the probable limiting constraint on ventilation is control of chemical contaminants. No information exists to adequately characterize the airborne chemical load in the hospital setting at the present time" (*26*).

Reduced air-exchange rates in the presence of emissions from building materials and consumer products may adversely affect human health, welfare, and comfort. Urea formaldehyde foam insulation (UFFI), for example, has been shown to be a significant source of formaldehyde in some instances (*27–29*). Complaints, symptoms, and illnesses have been reported by occupants of buildings with UFFI, and its use has been abandoned in Canada (*27*). The Consumer Products Safety Commission recently proposed a ban on UFFI in the United States (*28*); however, this action is still subject to legislative and judicial review.

Mobile homes and prefabricated housing units are especially prone to indoor air pollution problems. They have lower mean air-exchange rates than conventional homes, are of smaller volume, use proportionally more materials containing volatile organic resins, and are more likely to use propane for cooking fuel. There were 3,722,000 occupied mobile homes in the United States during 1977 (*10*) and about 250,000 new units will be shipped this year, an increase of 15 percent over the past 2 years (*30*). Mobile homes account for about 20 percent of the new housing market, and the number of mobile home occupants is expected to increase as restrictive zoning laws are phased out by many localities and states.

Another reason for concern is the growing number of building-related illnesses that have come to the attention of public health officials. The term "sick building syndrome" refers to health symptoms (for instance, irritation of mucous membranes, headaches, dizziness, nausea, diarrhea, rashes, and abdominal and chest pain) which affect occupants of a building. The Consumer Product Safety Commission has received more than 3000 complaints to date about exposures to contaminant releases from building materials (*29*). The National Institute for Occupational Safety and Health (NIOSH) conducted 115 investigations between mid-1978 and 1980 based on complaints from workers in nonindustrial settings who believed they were victims of building-related pollution (*31*). The New York City Department of Environmental Protection has received many complaints about indoor air pollution, despite the fact there is no established mechanism for receiving and dealing with consumer concerns about indoor air quality (*32*). Systematic investigations of building-associated illness are beginning (*31, 33*), but local, state, and federal agencies often lack explicit authority, funds, and expertise to deal with this issue.

In summary, there is growing evidence that evaluation of indoor as well as outdoor exposures to air pollution is essential for realistic health effects assessment. If indoor exposures are not taken into account in epidemiologic investigations of air pollution, systematic and random biases may give rise to spurious conclusions (*10, 34*). Total personal exposures are often better correlated with indoor than with outdoor concentrations (*35–38*). These findings, along with concerns about reduced air-exchange rates and new indoor pollution sources, challenge the premise that costly controls on sources of ambient pollution are improving public health through reduction of human exposures.

Indoor Pollutants and Sources

Several pollutants from indoor sources affect human health. Sidestream tobacco smoke, radon and radon decay products, asbestos fibers, fiber glass, formaldehyde, combustion by-products (such as polycyclic aromatic hydrocarbons, nitrogen dioxide, carbon monoxide, hydrogen cyanide, and sulfur dioxide), aeropathogens, and allergens are associated with a range of problems from mild irritation of nasal and mucous membranes to irreversible toxic and carcinogenic effects.

The available evidence of toxicity, indoor concentrations, and number of people exposed suggests that some indoor air pollutants may constitute significant public health problems. The more important pollutants identified to date are briefly reviewed below.

Combustion by-products. Indoor combustion of fuels can be a source of CO, CO_2, SO_2, formaldehyde, hydrocarbons, nitrogen oxides, and a variety of particles. Numerous studies have reported elevated indoor levels of NO_2, NO, CO, and CO_2 in homes with unvented appliances (*10*). Depending on source use and air-exchange rates, long-term indoor NO_2 averages can exceed the National Ambient Air Quality Standard (NAAQS) of 100 $\mu g/m^3$, even in areas where ambient outdoor values are 15 $\mu g/m^3$ (*34*). Peak hourly NO_2 concentrations between 200 and 700 $\mu g/m^3$ have been measured routinely in kitchens and other rooms of homes during conventional gas cooking. Elevated concentrations of NO_2 or CO have been reported for homes and schools where kerosene heaters and unvented gas heaters are used and in skating arenas with gasoline-powered ice-cleaning equipment (*28, 39*).

Exposures to NO_2 have been associated with toxicological effects including pulmonary edema, bronchoconstriction, and increased infection rates. Some epidemiologic evidence indicates that increased respiratory infection in young children and adult males and lower pulmonary function performance are associated with a history of exposure to gas stove emissions. Other studies failed to show significant effects associated with gas cooking (*40*).

Carbon monoxide from faulty furnaces and attached garages is responsible for several fatal accidents each year. Under typical use conditions, emissions from cooking appliances may raise short-term indoor CO concentrations 5 to 10 ppm. When a gas stove is used for heating, a common practice among urban poor in northern climates, concentrations of 25 to 50 ppm have been measured (*41*). Concentrations ranging from 25 to more than 100 ppm have been measured in ice-skating rinks, apartments, and offices with attached or underground garages.

Carbon monoxide forms carboxyhemoglobin (COHb) in the blood and inhibits oxygen uptake. It is not known at present whether there is a threshold for adverse effects from oxygen deprivation due to COHb. Recent work indicates that exercising adults with angina pectoris are sensitive to COHb concentrations as low as 1 percent (*42*). Community air

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pollution and indoor exposures to combustion by-products or sidestream cigarette smoke can raise COHb in nonsmokers to 2 to 3 percent (*43*).

National Ambient Air Quality Standards have been promulgated for CO and NO_2, and emission controls for mobile and stationary sources are required by legislative mandate. A limited number of personal exposure studies showed significant correlations between exposures and indoor concentrations. The evidence suggests that many, if not most, exposures to NO_2 and CO occur indoors and are therefore not represented accurately by outdoor monitors (*10, 34, 44*).

Tobacco smoke. Tobacco smoke is discussed separately for several reasons. First, nearly everyone is exposed at one time or another to tobacco smoke. Although 33 percent of the adult population regularly smoke cigarettes, this figure does not necessarily characterize the population of exposed children. For example, epidemiologic studies in the United States have shown that the percentage of children living in homes with one or more smokers ranged from 54 in Tucson to 76 for a middle-income community in St. Louis (*10*). Second, more than 2000 compounds have been identified in cigarette smoke, many of which are established carcinogens, irritants, and asphyxiants. And third, there is increasing evidence that passive exposures to tobacco smoke may affect respiratory health.

Tobacco combustion indoors contributes to concentrations of respirable particles, nicotine, polycyclic aromatic hydrocarbons, CO, acrolein, NO_2, and many other substances. The resulting concentrations vary widely, depending on the frequency and amount of smoking, air-infiltration rates, air-cleaning devices, and air-distribution systems. Measurements in bars, restaurants, airplanes (smoking section), buses, conference rooms, and offices indicate CO and particulate matter concentrations of 2 to 35 ppm and 10 to 1000 $\mu g/m^3$, respectively (*45*). Analysis of respirable particle samples collected in 80 homes over several years indicates that a "pack-a-day" smoker will contribute approximately 20 $\mu g/m^3$ to 24-hour indoor particle concentrations (*46*). Where two or more heavy smokers resided in a house with low air-exchange rates, the 24-hour NAAQS of 260 $\mu g/m^3$ was also violated.

While the health effects of smoking on smokers have been studied extensively (*47*), the health effects on nonsmokers have received far less emphasis. Many substances in cigarette smoke are irritants, and conjunctival irritation, nasal discomfort, cough, sore throat, and sneezing have been noted in nonsmokers exposed to cigarette smoke (*48*). Measured changes in heart rate, systolic blood pressure, COHb, psychomotor functions, and small airway dysfunction have also been reported in nonsmokers who are exposed to smoke (*43, 49–51*).

Most studies of the effect of parental smoking on respiratory health in children show an association with reported respiratory morbidity in children, especially

Table 1. Summary of indoor pollutants, emission sources, and concentrations [adapted from (*10*)]. Column 3 shows typical ranges of indoor concentrations in the presence of indoor emission sources.

Pollutant	Major emission sources	Typical indoor concentrations	Indoor/outdoor concentration ratio
	Origin: predominantly outdoors		
Sulfur oxides (gases, particles)	Fuel combustion, smelters	0–15 $\mu g/m^3$	< 1
Ozone	Photochemical reactions	0–10 ppb	< 1
Pollens	Trees, grass, weeds, plants	L.V.*	< 1
Lead, manganese	Automobiles	L.V.	< 1
Calcium, chlorine, silicon, cadmium	Suspension of soils, industrial emissions	N.A.†	< 1
Organic substances	Petrochemical solvents, natural sources, vaporization of unburned fuels	N.A.	< 1
	Origin: indoors or outdoors		
Nitric oxide, nitrogen dioxide	Fuel burning	10–120 $\mu g/m^3$‡ 200–700 $\mu g/m^3$§	> 1
Carbon monoxide	Fuel burning	5–50 ppm	> 1
Carbon dioxide	Metabolic activity, combustion	2000–3000 ppm	> 1
Particles	Resuspension, condensation of vapors, combustion products	10–1000 $\mu g/m^3$	1
Water vapor	Biological activity, combustion evaporation	N.A.	> 1
Organic substances	Volatilization, combustion, paint, metabolic action, pesticides	N.A.	> 1
Spores	Fungi, molds	N.A.	> 1
	Origin: predominantly indoors		
Radon	Building construction materials (concrete, stone), water	0.01–4 pCi/liter	> 1
Formaldehyde	Particleboard, insulation, furnishings, tobacco smoke	0.01–0.5 ppm	> 1
Asbestos, mineral, and synthetic fibers	Fire retardant materials, insulation	0–1 fiber/ml	1
Organic substances	Adhesives, solvents, cooking, cosmetics	L.V.	> 1
Ammonia	Metabolic activity, cleaning products	N.A.	> 1
Polycyclic hydrocarbons, arsenic, nicotine, acrolein, and so forth	Tobacco smoke	L.V.	> 1
Mercury	Fungicides, paints, spills in dental-care facilities or labs, thermometer breakage	L.V.	> 1
Aerosols	Consumer products	N.A.	> 1
Microorganisms	People, animals, plants	L.V.	> 1
Allergens	House dust, animal dander, insect parts	L.V.	> 1

*L.V., limited and variable (limited measurements, high variation). †N.A., not applicable. ‡Annual average. §One-hour average in homes with gas stoves, during cooking.

during the first 2 years of life (*10, 47, 52*). An association between the number of cigarettes smoked per day by the parents and respiratory symptoms in children has been a consistent finding. However, these relations do not always appear independent of parental symptoms, socioeconomic class, or smoking habits of the children. Some investigators found an association between tobacco smoke exposure of nonsmoking wives whose husbands smoke and increased lung cancer rates (*53–55*).

The National Academy of Sciences stated in its report on indoor pollutants (*10*): "The constituents of tobacco smoke are well-documented as hazardous, the prevalence of population exposure is very high, and there is an increased incidence of respiratory tract symptoms and functional decrements (decreases) in children residing in homes with smokers, compared with those homes without smokers. These considerations and recent evidence of increased lung cancer rates among non-smoking women living with smoking husbands have led us to conclude that indoor exposure to tobacco smoke has adverse effects. Public policy should clearly articulate that involuntary exposure to tobacco smoke ought to be minimized or avoided where possible."

Radon and radon decay products. Radon is a radioactive decay product of radium-226. Radium, a natural trace constituent of rock and soil, is found in building materials made from earth crustal components. Radon-222 is a gas with a half-life of 3.8 days. It decays through polonium-218, lead-214, bismuth-214, and polonium-214 before reaching lead-206, a stable isotope. These decay products are solids and can attach to aerosols, which may become embedded in the lungs and irradiate surrounding tissue. It is usually radon gas which diffuses into indoor air from the ground, building materials, or well water. Higher concentrations are typically measured in basements, crawl spaces, and homes with low air-exchange rates.

Radon and radon decay product concentrations have been measured in conventional and experimental homes in several states (*10, 56*). Typical radon concentrations range from 0.01 to 4 pCi/liter. In "energy-efficient" houses, levels exceeding 20 pCi/liter have been reported. Concentrations ranging from 0.1 to 27 pCi/liter were measured in 52 conventional houses in Maryland (*57*). Integrated samples during the winter in 4000 Swedish homes averaged about three times the concentrations reported for Maine homes and 20 to 30 times the concentrations reported for homes in California and Texas (*58*).

Concerns about adverse health effects of indoor exposure to radon decay products are based on the higher incidence of lung cancer observed in uranium miners. Although indoor home exposures are thought to be considerably lower, risk is proportional to exposure and the number of people exposed is large. Recent reviews of epidemiologic studies of radon and lung cancer in miners estimate that lifetime risks range from 21 to 54 (*59*) to 1000 (*59, 60*) deaths per 10^6 working level months (WLM) (*61*). For the range of in-home radon exposures typically recorded, 0.04 to 0.8 WLM, excess lung cancers in the United States have been estimated to be as high as 10,000 (*56*).

Microorganisms and allergens. A large variety of biological material is present in indoor environments. Inhalation of biological aerosols discharged by people and animals is a primary mechanism of contagion for most acute respiratory infections (*62*). Tuberculosis, measles, smallpox, and staphylococci are known to be transmitted by air ventilation systems in schools and hospitals. Air-cooling equipment, cool-mist vaporizers, humidifiers, nebulizers, flush toilets, ice machines, and carpeting can incubate and distribute bacterial aerosols indoors. Legionnaire's disease (*Legionella pneumophila*) and humidifier fever are well-known examples of air-conditioning–related bacteria (*63, 64*).

According to the National Health Survey (*65*), respiratory ailments (predominantly upper respiratory disease and influenza) account for more than half of all acute conditions, including illnesses and injuries. The incidence of respiratory conditions is just under one per person per year, and they typically restrict activity for 4.5 days. Higher incidences are observed among younger children and the elderly. Considering the loss of time from work and school, as well as medical costs, the impact of indoor contagion is probably enormous (*66*). There is a lack of data with which to evaluate the relation between infection and ventilation rates. If the primary mechanism is by contact rather than inhalation, the prevalence of respiratory infection may be unaffected by changes in ventilation.

Pollen, molds, mites, chemical additives, animal dander, fungi, algae, and insect parts are known indoor allergens. Sources of indoor allergenic materials include pets, detergents, humidifier and air-cooling fluids, growth of molds and fungi on surfaces, and insects that live in dust and vents. Temperature and humidity conditions are important for many indoor aeroallergens. For example, house mites flourish at temperatures around 25°C and relative humidities above 45 percent. Because high humidity favors the growth of molds and fungi, tightly sealed buildings in humid climates are more prone to allergenic problems.

Reduced ventilation and increased use of untreated recirculated air may increase concentrations of microorganisms. Prolonged exposure to some chemicals and antigens can cause sensitization. Therefore, reduced fresh air in buildings might lead to increased rates of infection and allergy. However, little is known about sources, concentrations, and survival rates of many aeropathogens indoors.

Formaldehyde and other organic compounds. Building materials (plywood, particleboard, and so on), furnishings (carpets, draperies), and some types of foam insulation contain formaldehyde resins, the most common of which is urea formaldehyde. Excess formaldehyde in these products can be released over a considerable period. Outgassing rates are higher for new materials and are directly influenced by humidity and temperature. Although few longitudinal studies have been done, the half-life for formaldehyde emissions is approximately 4.4 years. Unvented gas combustion and tobacco smoking are other sources of indoor formaldehyde.

Indoor formaldehyde sampling has been conducted principally in locations where higher concentrations were suspected. Measurements in Denmark, the Netherlands, the Federal Republic of Germany, Sweden, and the United States have shown that formaldehyde concentrations often exceed 0.1 ppm. In 23 Danish homes the average formaldehyde concentration was 0.5 ppm and the range was 0.07 to 1.9 ppm (*67*). In response to occupant complaints, formaldehyde concentrations in more than 200 mobile homes in Washington State were measured and were found to range from 0.03 to 2.4 ppm (*68*). Similar findings were reported for mobile homes in Minnesota and Wisconsin (*69*). Formaldehyde concentrations of 0.1 to 0.5 ppm have been measured in conventional homes and schools without obvious sources. Concentrations in excess of 0.1 ppm appear to be common in homes insulated with UFFI.

Adverse effects from formaldehyde may result from inhalation, ingestion, or contact. The compound showed mutagenic activity in a variety of microorganisms and produced nasopharyngeal carcinoma in laboratory rats and mice (*68*). Chamber studies with humans showed

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Table 2. Control measures for indoor air pollutants.

Control measure description	Pollutant	Example
Ventilation: Dilution of indoor air with fresh outdoor air or recirculated filtered air, using mechanical or natural methods to promote localized, zonal, or general ventilation	Radon and radon progeny; combustion by-products; tobacco smoke; biological agents (particles)	Local exhaust of gas stove emissions; air-to-air heat exchangers; building ventilation codes
Source removal or substitution: Removal of indoor emission sources or substitution of less hazardous materials or products	Organic substances; asbestiform minerals; tobacco smoke	Restrictions on smoking in public places; removal of asbestos
Some modification: Reduction of emission rates through changes in design or processes; containment of emissions by barriers or sealants	Radon and radon progeny; organic substances; asbestiform minerals; combustion by-products	Plastic barriers to reduce radon levels; containment of asbestos; design of buildings without basements to avoid radon; catalytic oxidation of CO to CO_2 in kerosene burners
Air cleaning: Purification of indoor air by gas adsorbers, air filters, and electrostatic precipitators	Particulate matter; combustion by-products; biological agents (particles)	Residential air cleaners to control tobacco smoke or wood smoke; ultraviolet irradiation to decontaminate ventilation air; formaldehyde sorbant filters
Behavioral adjustment: Reduction in human exposure through modification of behavior patterns; facilitated by consumer education, product labeling, building design, warning devices, and legal liability	Organic substances; combustion by-products; tobacco smoke	Smoke-free zones; architectural design of interior space; certification of formaldehyde concentrations for home purchases

eye discomfort in the concentration range 0.1 to 0.4 ppm (68), while residential exposures as low as 0.02 ppm were associated with tearing and eye irritation. The fact that in-home responses occur at lower exposures may reflect a broader spectrum of sensitivity in the population at large, increased sensitivity due to prolonged low-level exposures, adaptation of volunteers in the chamber studies to elevated formaldehyde concentrations, or effects of other irritants present in indoor air. At concentrations of 10 to 100 ppm, formaldehyde exposures can cause lower respiratory irritation and pulmonary edema.

Formaldehyde effects on the nervous system are not well understood, although psychological and neurophysical effects have been reported (*10, 70*). The results are difficult to interpret, but suggest that formaldehyde levels of ~1 ppm can affect the central nervous system. Effects include subtle changes such as short-term memory loss, increased anxiety, and slight changes in adaptation to darkness.

A variety of other organic contaminants have also been identified in indoor environments. People emit bioeffluents such as acetone, butyric acid, ethyl and methyl alcohol, and other acids and alcohols. Combustion of wood, kerosene, and tobacco produces polycyclic aromatic hydrocarbons. Application of pesticides can release chlorinated hydrocarbons or organophosphate compounds. Personal care products, cleaning materials, paints, lacquers, and varnishes generate chlorinated compounds, acetone, ammonia, toluene, and benzene. Health risks associated with indoor exposures to this diverse group of chemicals have not been investigated adequately.

Asbestos fibers. Because of the widespread use of asbestos-containing products in ceiling tiles, floor tiles, pipe insulation, spackling compounds, concrete, and acoustical and thermal insulating material, there is a large potential for public exposure (*71, 72*). Acute exposures to asbestos and glass fibers cause severe skin irritation. A variety of neoplastic diseases, with latency periods of 15 or more years, have been associated with asbestos exposures. Increases in lung cancers, pleural and peritoneal mesotheliomas, and gastrointestinal tract cancers have been linked to occupational exposures (*73–75*). Several studies have shown increased mesothelioma rates among persons living near asbestos-production facilities and shipyards and among family members living with asbestos-exposed workers (*76–79*).

Airborne fiber concentrations determined by NIOSH standard methods range from zero during normal activities outdoors and in schools, offices, and dormitories to more than 100 fibers per milliliter during disrupting contact maintenance to ceilings (*76, 80, 81*). Unless the material is extremely friable, which is rare, asbestos exposures are likely to be episodic and therefore difficult to evaluate by random monitoring. Electron microscopic examination of air samples revealed values ranging from nearly zero outdoors in many U.S. cities to about 2000 ng/m^3 in a New Jersey school during custodial activities (*72, 80, 81*). Current asbestos standards include an Occupational Safety and Health Administration (OSHA) standard of 2 fibers per milliliter (*73, 82*) and a recommended NIOSH limit of 0.1 fiber per milliliter (*83*).

Although few measurements of air concentrations are available, it is clear that houses, schools, and office buildings can become contaminated. Risks to the general public are unknown since only exposure-effect data from occupational settings are available. However, the possible synergism with other contaminants, particularly cigarette smoke, and the severity of the potential health consequences justify steps to limit exposure to asbestos.

Most (85 percent or more) of the asbestos in use is immobilized in strong binding material (*84*). However, if this material is deliberately or accidentally disrupted, asbestos fibers can be released. Because the material can be found in easily accessible places and in ventilation systems, there is a potential for fiber release as a result of maintenance, renovation, negligence, or vandalism. In 1978, the U.S. Environmntal Protection Agency (EPA) banned spray-on application of asbestos-containing substances, except those in which the asbestos fibers are encapsulated with a bituminous or resinous binder during spraying to reduce friability after drying (*85*).

Controls

Workable and effective control strategies must be based on an understanding of several pertinent factors. First, contaminant characteristics need to be assessed, including: concentrations, reac-

tivity, physical state, and particle size, if applicable. Second, emission source configurations should be taken into account. Are discharges continuous or intermittent, are they point or area releases, and do they originate primarily indoors or outdoors? Third, the nature of exposure response relations must be considered. Are individuals to be protected from long-term exposures to low concentrations or periodic short-term exposures to peak concentrations? Finally, the type of indoor enclosure is important. Some ameliorating measures are more suited to private residences than to public buildings, or to new, as opposed to existing, structures.

Identified indoor pollution control methods fall into five general categories: ventilation; source removal or substitution; source modification; air purification; and behavioral adjustments to reduce exposures (avoidance). These classifications are not mutually exclusive and effectual strategies might use combinations, such as ventilation, source removal, and behavioral changes to reduce nonvoluntary exposures to tobacco smoke. Table 2 summarizes the applicability of control methods to important indoor air contaminants.

Public Policy Issues

Realization that indoor air pollution may represent a public health hazard presents policy-makers with familiar questions: Is the problem serious enough to warrant official intervention and, if so, what public actions are most appropriate? Healthful indoor air quality—what it is and how to achieve it—is not yet well understood, and scientific data on exposures and associated health effects are lacking. Nevertheless, the mounting evidence of elevated indoor contaminant levels suggests that government efforts to safeguard citizens' health and safety may be justified.

Government response so far has been piecemeal and complaint-oriented. In some cases, such as asbestos, many different federal agencies have jurisdiction over portions of the problem. For other pollutants, like microorganisms, combustion by-products, and organic emissions from building materials, responsibility is not clearly defined. No overall strategy exists to provide a coordinated, well-managed approach to ensure adequate indoor air quality.

Historically, government measures to redress environmental pollution have been taken when identified pollutant hazards receive abundant attention in the media and sufficient public awareness is generated. With the possible exception of antismoking groups, no organized constituency has developed to champion the cause of clean air inside buildings. The absence of a group lobbying for healthful indoor air quality removes much of the political urgency normally associated with environmental problems. As results of ongoing research become available, it is likely that public pressure for official intervention will build and legislators will have more incentive to take action.

A more structured and institutionalized approach is advocated by many professionals involved with this issue. Suggestions have included: coordinated interagency research (*86*); consolidation of federal responsibility within one agency (*6, 87, 88*); amendment of the Clean Air Act (*6, 11*); and granting of authority to states to regulate indoor air quality (*89*). A bill (HR-6323) was introduced in Congress during 1982 to appropriate funds for the Environmental Protection Agency to study the problem and recommend remedial actions.

As Sexton and Repetto (*90*) pointed out, it is essential to realize that a fundamental difference exists between indoor and outdoor air. Outdoor air is a "public good" in the sense that members of a community breathe basically the same ambient air. No rational individual would undertake the task of cleaning up the air over Boston, for example, since his or her share of the benefits would be much smaller than the costs. Nor would voluntary cooperation suffice, since those who refused to contribute could not be excluded from enjoying the benefits of reduced pollution. Similarly, in the absence of regulations or legal liability, no pollution source would spend enough on abatement, due to the difficulty of collecting from beneficiaries. The rationale for government regulation of outdoor air pollution has focused on the issue that those who suffer the effects are not compensated, nor is their interest in cleaner air readily effective in influencing polluters.

The situation is quite different for some indoor environments, especially private residences. Both the costs and benefits of pollution control are internalized within households. If occupants foul the air in their own home, they are forced to breathe it. If they attempt to improve its quality by increasing ventilation or installing air-cleaning devices, they bear the costs and enjoy the benefits. For some contaminants, such as tobacco smoke, odorants, and water vapor, benefits are readily recognizable through improvements in perceptible air quality and reduction of corrosion, soiling, and molds. For pollutants that are harmful to human health, but below perception thresholds, benefits will include reduced health risks due to lower exposures.

Creation of a regulatory framework for indoor air quality poses special policy issues which bear directly on choices about appropriate public responses (*90*). Certain aspects of nonworkplace environments are now subject to government ordinances, including residential and commercial building codes, health regulations, safety rules, and fire ordinances. However, this form of intervention is not necessarily optimal or even desirable. Although there are similarities between indoor and outdoor air, the complex set of regulations comprising the Clean Air Act should not automatically serve as a guide for indoor control strategies. Setting strict indoor air quality standards would almost certainly be expensive because of the costs associated with monitoring and regulating approximately 100 million buildings in the United States. Perhaps the most serious impediments to the regulatory approach are public antipathy toward this form of intervention and problems associated with enforcement. The diversity of nonoccupational indoor environments needs to be considered before practical and cost-effective control strategies can be designed and implemented.

It must also be taken into account that households are already making decisions about their own air quality. Promulgation of regulations might or might not improve those decisions. To determine the appropriateness of regulatory intervention it is necessary to do more than compile information about pollutant concentrations, human exposures, and associated health hazards. It is equally important to obtain information about individual perceptions of indoor air quality, public awareness of health risks, and the extent to which better information influences consumer choices. Commonsense precautions by building occupants may well prove to be the most effective and least expensive control measures. For this reason, the effectiveness of public information programs in promoting behavioral adjustments that will reduce personal exposures to air pollution needs to be evaluated (for instance, voluntary segregation of smokers and nonsmokers inside buildings).

In the development of effective public policy, the responsibilities of different sectors in society that have a stake in this issue should be emphasized. There

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are important distinctions between the responsibilities of individuals, building designers, contractors, operators and owners, professional organizations, product and material manufacturers, and government. Major responsibilities that should be formally recognized are summarized in Table 3.

Recommendations

The issue of indoor air pollution and its effect on public health is complex. Yet because society has been slow to recognize the importance of healthful indoor air quality, the information we have is fragmented, anecdotal, and often contradictory. As with other environmental contaminants, strategies for reducing risk to exposed populations must be based on defensible exposure-response relations. In this way, the cost-effectiveness of various control options can be evaluated on the basis of reductions in population exposures. Table 4 shows the important components of a comprehensive evaluation of indoor air pollution and summarizes our current knowledge.

Several major issues are involved in decisions concerning the need for public action to deal with indoor air pollution. First, the role of government may depend on the degree of "publicness" of a particular building. The rationale for government intervention is stronger for public buildings (such as hospitals) than private residences. Government responsibility may be different for occupational and nonoccupational settings and for existing and planned buildings.

Second, dissimilarities between indoor emission sources may be important. Many contaminants in indoor environments (tobacco smoke, combustion byproducts) result from human activities, while others (radon, formaldehyde) are less dependent on the activity patterns of occupants. Behavioral adjustment (such as prohibiting smoking in public places or using hood ventilation during cooking) may be the most effective and inexpensive way to control pollutants arising from discretionary actions. For pollutants not directly related to human activities, such as those emitted by soil, tap water, building materials, and furnishings, stricter building codes, simple air-cleaning devices, or sealants might be required.

Third, some indoor pollutants, such as tobacco smoke, are perceptible to most people, and individual actions to reduce personal exposures may be predicated on sensory stimuli. Other pollutants are

Table 3. Responsibilities for healthful indoor environments.

Individuals

Maintain and properly use products and appliances
Exercise direct discretionary control of ventilation in most residential and some occupational circumstances

Building owners or managers

Operate and maintain a balanced ventilation system in compliance with building ventilation codes
Use zone ventilation or local exhaust for indoor contaminant sources
Properly use cleaning solvents, paints, varnishes, herbicides, insecticides, furnishings, and insulation

Architects, developers, contractors

Adopt protection of indoor air quality as a design objective
Design ventilation systems to comply with new ASHRAE standard 62-1981
Provide for separation of occupants and indoor pollutant sources
Elimination or containment of potential sources

Manufacturers

Test, certify, and label products that are potential air pollution sources
Conduct research on potential health and comfort effects resulting from normal use or possible misuse of products
Substitute less harmful products and materials, if necessary

Government

Ensure healthfulness of indoor environments built, maintained, supervised, or financed through public funding
Ensure compliance with building ventilation codes and acceptable indoor air quality throughout the occupied life of a building
Sponsor research to assess indoor concentrations, health and comfort effects, control and policy options
Establish model or mandatory guidelines, codes, ordinances, or performance standards to protect the public
Provide information and assistance to state and local governments
Advise the public on safety of products, construction materials and practices, availability of monitoring equipment, and performance of HVAC systems

Table 4. Components of comprehensive evaluation of indoor air pollution and state of current knowledge.

Emission sources

Chamber studies done for several sources
Few measurements under dynamic conditions
Few studies of emission rates during normal use
Lack of information about distribution of sources within population

Dilution

Understanding of basic components affecting air-exchange rates
Measurement techniques available
Site-specific models developed, but more general application problematic
Only limited information available on distribution of air-exchange rates in existing buildings
Mixing inside buildings without mechanical ventilation systems not well understood

Indoor concentrations

Survey-type data collected for some pollutants
Applicability of survey data to entire building stock unknown
Dilution and mechanical filtration typically assumed to be first-order determinants of concentrations
Chemical and physical interactions, as well as removal rates, not well defined
Little known about variations in both removal and penetration rate

Human activity patterns

General features of population activity patterns known
Insufficient information about variations in activity patterns with age, sex, socioeconomic class, employment status, location, and season

Exposures

Relatively few studies of personal exposures to air pollution
Limited data indicate poor correlation between outdoor concentrations and personal exposures
Lack of suitable instrumentation limits application of personal exposure studies
Distribution of exposures across the population and effects of energy conservation on indoor exposures not known

Health effects

Irritant, toxic, mutagenic, and carcinogenic effects noted for many indoor contaminants
Additional information needed on central nervous system effects
Epidemiologic evidence of adverse health effects available for some pollutants
Data on dose-response relation accumulated for a few pollutants
Numbers, characteristics, and distribution of chemically sensitive individuals not known
Information lacking on health effects of long-term, integrated exposures compared to short-term, peak exposures

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below perception thresholds (radon, microorganisms, asbestos, CO, NO_2, and so on). Consideration of voluntary and nonvoluntary risks is important for policy decisions. Development of simple warning devices could provide individuals with information on which to base decisions about the appropriateness of remedial measures. Such actions could reduce the need for government intervention to mitigate public health risks.

Fourth, it is necessary to specify whether building occupants are to be protected from chronic exposures to low levels of pollution or short-term peak exposures. If long-term exposures are considered important, then reductions in total human exposures to air pollution should be the goal. If short-term exposures are critical, then efforts should be focused on identifying peak concentrations and protecting the population at risk (for instance, limiting CO exposures for building occupants who use gas-cooking stoves for space heating).

Fifth, policy-makers must balance the benefits of energy conservation measures (for instance, reduced ambient pollution from fossil-fueled power plants) against the costs of deteriorating indoor air quality. Determining the essential components of healthful indoor air is a fundamental part of this process.

Sixth, if it is decided that public intervention is needed, a regulatory approach should not automatically be adopted. Policy alternatives such as economic incentives, better definition of legal rights and liabilities, public information programs, and expanded administrative efforts based on existing legislation might be more appropriate for control of indoor environmental hazards.

Because data from several studies indicate indoor exposures to some pollutants may represent significant health risks, official efforts to define the magnitude and extent of public health consequences seem justified. However, the future of public efforts to deal with indoor air pollution in nonoccupational environments is uncertain. Adequate funding is not currently available. Given the present regulatory climate of limited government intrusion and increasing reliance on free-market economics, it seems unlikely that programs focusing on indoor air pollution will be initiated in the near future. We believe that an overall strategy should be developed to ensure a coordinated, well-managed investigation of indoor air pollution exposures and their health consequences. In combination with efforts to define the problem, control options and policy alternatives should be evaluated.

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State of Art

Health Effects and Sources of Indoor Air Pollution. Part I[1-3]

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Contents

SUMMARY Since the early 1970s, the health effects of indoor air pollution have been investigated with increasing intensity. Consequently, a large body of literature is now available on diverse aspects of indoor air pollution: sources, concentrations, health effects, engineering, and policy. This review begins with a review of the principal pollutants found in indoor environments and their sources. Subsequently, exposure to indoor air pollutants and health effects are considered, with an emphasis on those indoor air quality problems of greatest concern at present: passive exposure to tobacco smoke, nitrogen dioxide from gas-fueled cooking stoves, formaldehyde exposure, radon daughter exposure, and the diverse health problems encountered by workers in newer sealed office buildings. The review concludes by briefly addressing assessment of indoor air quality, control technology, research needs, and clinical implications.

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Introduction

During this century, dramatic episodes of excess mortality caused by ambient air pollution convincingly established that atmospheric contamination by human activities can adversely affect health. In many countries, governmental regulations implemented in response to the adverse health effects of air pollution have resulted in strong trends towards improved air quality. As the hazards posed by ambient air pollution from conventional fossil fuels have diminished in some countries, the relevance of indoor air quality for health has become increasingly apparent. Studies of time-activity patterns demonstrate that residents of more developed countries spend on average little time outdoors (table 1) (1, 2). Thus, indoor pollutant concentrations are the predominant determinant of exposure for many pollutants and the only source of exposure for some. However, pollutants in outdoor air do penetrate indoors, and for some pollutants of current importance, such as ozone and acid aerosols, nearly all exposure, whether received indoors or outdoors, results from outdoor sources.

Research directed at indoor air pollution and its adverse health effects began in the late 1960s and early 1970s (3, 4). Investigation in this area was subsequently stimulated by concerns that reduced ventilation of buildings for the purpose of energy conservation would increase pollutant concentrations and lead to adverse effects on health. Consequently, a large body of literature is now available on diverse aspects of indoor air pollution: sources, concentrations, health effects, mitigation, and policy.

While many health effects of indoor air pollution remain controversial, epidemiologic and clinical research has identified some health effects that should be considered by chest physicians and other health care providers. The public has been intensely interested in the new information on indoor air pollution, particularly as it relates to such ubiquitous exposures as formaldehyde, environmental tobacco smoke, radon and radon daughters, nitrogen dioxide (NO_2) from gas-fueled cooking stoves, and smoke from woodburning fireplaces and stoves. Patients may turn to their health care

This is Part I of two parts; the second will appear in the next issue of the Review.

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TABLE 1

AVERAGE HOURS SPENT PER DAY IN VARIOUS LOCATIONS BY ADULTS IN 44 U.S. CITIES*

Location	Employed Men	Employed Women	Housewives
At home	13.4 (55.8)†	15.4 (64.2)	20.5 (85.4)
At work	6.7 (27.9)	5.2 (21.7)	— (0)
In transit	1.6 (6.7)	1.3 (5 4)	1.0 (4.2)
Outside	0.7 (2.9)	0.3 (1.3)	0.4 (1.7)
Inside other structures	1.6 (6.7)	1.8 (7.5)	2.1 (8.8)

* Based on data in tables 7-1.1 and 7-1.3, page 795 in reference 1. Time calculated for "outside" includes the categories "just outside one's home" and "in all other locations." Time calculated for "inside other structures" consists of the categories "in other people's homes," "in places of business," and "in restaurants and bars." The original data did not separate work into indoor and outdoor categories.

† Percentage of 24 h

providers because of concerns about potential health effects of these and other indoor air pollutants.

The findings reported in this new literature also have wide-ranging policy implications (5). Evidence of adverse health effects of indoor air quality may require decisions and actions on consumer products, building materials and design, energy conservation practices, and regulation of smoking in public places. In the United States, the Environmental Protection Agency under the authority of the Clean Air Act regulates to protect and enhance outdoor but not indoor air quality. This agency has constructed a statutory framework for implementing ambient air quality standards and has devised a complex set of regulations for controlling mobile and stationary air pollution sources.

For some criteria pollutants, an encouraging trend of improving outdoor air quality has resulted. The number of locations exceeding the primary National Ambient Air Quality Standards for total suspended particles (TSP), sulfur dioxide (SO_2), carbon monoxide (CO), and lead (Pb) has decreased over the past decade. Even peak ozone (O_3) concentrations have declined in many locations. Of the 6 criteria pollutants, only NO_2 pollution has worsened (6). However, improvements in ambient air quality do not necessarily imply that human exposures to harmful pollutants have also declined. Indoor air quality is not directly regulated, and use of some sources of indoor air pollution, such as wood stoves and kerosene space heaters, is increasingly widespread. Low air exchange rates in newer homes and office buildings may also increase personal exposures. Thus, air quality policy designed to fully protect public health must address exposures to pollutants indoors as well as outdoors.

This review summarizes information on the health effects of indoor air pollution, with an emphasis on the data that are most relevant for health care providers and those concerned with public health aspects of indoor air quality. We have also focused on the indoor air quality problems of greatest public health concern at present and emphasize those for which new evidence has become available: passive exposure to tobacco smoke, NO_2 exposure from gas-fueled cooking stoves, formaldehyde exposure, radon daughter exposure, and the diverse health problems encountered by workers in newer sealed office buildings. The citations are based primarily on a literature search that extended through June 1986; selected references subsequent to that date have been cited.

Review articles (7–10) and several monographs (11–14) on indoor air pollution have been published, as well as a report by the National Research Council (15). Proceedings of meetings on this topic have also been published (16–24). Numerous sources on the health effects of ambient air pollution are also available, including a statement of the American Thoracic Society (25), reports on individual pollutants by the National Research Council, and the criteria documents prepared periodically by the Environmental Protection Agency.

In this review, we initially consider the sources of indoor air pollution and information on personal exposures to indoor air pollution. Subsequently, for each of the major pollutants, we review the concentrations in indoor environments and the health effects. We conclude by briefly addressing indoor air quality assessment, control technology, research needs, and clinical implications.

Indoor Air Pollution: Sources and Exposure

Introduction

In this section, we highlight information on the sources of those pollutants that have been, or are, potentially associated with disease. We also review studies of personal exposures to pollutants. Concentrations of pollutants in indoor environments are described in subsequent sections on individual pollutants. We do not attempt to cover exhaustively the data on sources and exposures; comprehensive treatments are available in the report of the National Research Council (15) on indoor air pollution, in a review by Yocom (26), and in the proceedings of the Seventh Oak Ridge National Laboratory Life Sciences Symposium (24).

The health risks posed by air pollution are determined by the personal exposure of individuals to contaminants and not simply by pollutant concentrations in indoor and outdoor air. Personal exposures to air pollutants represent the average of the pollutant concentrations encountered in various environments with weighting proportional to the time spent in each location (figure 1). In more developed countries, studies of activity patterns have established the importance of the indoor environment in determining personal exposures (table 1) (1, 2).

The determinants of indoor concentrations vary among the pollutants. Levels may be influenced by outdoor levels, indoor sources, the rate of exchange between indoor and outdoor air, and other characteristics of the structure and its furnishings that influence pollutant dispersion and removal (15). Pollutants from

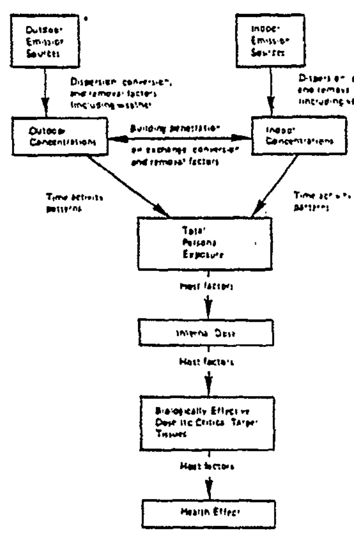

Fig. 1. Framework for considering the relationships among pollutant concentrations, personal exposures, doses of pollutants to target tissues, and health effects (Reprinted with permission from reference 27.)

outdoor sources can enter a building through mechanical ventilation systems and through the natural infiltration of air. The indoor concentrations of "outdoor pollutants" depend not only upon the outdoor concentrations but upon the rate of air infiltration, the reactivity of the contaminant, the efficiency of any mechanical filtration systems, and, for solids, upon the particle size and shape. For example, O_3, primarily an ambient pollutant, is a highly reactive molecule; it follows first-order decay kinetics when penetrating indoors and usually reacts quickly with surfaces. Indoor concentrations of O_3 are usually less than 50% of outdoor concentrations (28). Nevertheless, even small amounts of O_3 will be important in some indoor environments, such as art museums and locations with NO_2 sources. The concentrations of pollens, which are large particles from outside sources, decline substantially with increasing distance from doors and windows (29). Outdoor sources may lead to unusually high pollutant levels indoors if fresh air intake vents are improperly located. For example, increased CO levels have been measured in buildings with intake vents fed by air contaminated with vehicle exhaust from adjacent roadways or parking garages.

TABLE 2

TYPICAL SOURCES OF INDOOR AIR POLLUTION IN THE HOME, OFFICE, AND TRANSPORTATION ENVIRONMENT

Environment	Source and Pollutants
Home	Tobacco smoking: respirable particles, CO, VOC*
	Gas stoves: NO_2, CO
	Woodstoves and fireplaces; respirable particles, CO, PAH†
	Building materials: formaldehyde, radon
	Earth underlying the home: radon
	Furnishings and household products: VOC, formaldehyde
	Gas-fueled space heaters: NO_2, CO
	Kerosene-fueled space heaters: NO_2, CO, SO_2
	Insulation: asbestos
	Moist materials and surfaces: biological agents
Office	Tobacco smoking: respirable particles, CO, VOC
	Building materials: VOC, formaldehyde
	Furnishings: VOC, formaldehyde
	Copying machines: VOC
	Air conditioning systems: biological agents, vehicle exhaust with combustion emissions containing particles, CO, and NO_2
Transportation	Tobacco smoking: respirable particles, CO, VOC
	Ambient air: ozone in jet aircraft, CO and lead in automobiles
	Auto air conditioners: biological agents

* Volatile organic compounds
† Polycyclic aromatic hydrocarbons

Increasing concentrations of indoor air pollutants have been of particular concern as ventilation rates have been reduced in newer structures. The 83 million housing units in the United States are diverse in character and some types are more likely to be associated with excessive indoor air pollution than are others. In new "tight" homes, air exchange rates during winter may average less than 0.5 per hour, while most conventional homes have average exchange rates between indoor and outdoor air around 1.0 per hour. Energy efficient "super tight" homes can be built with winter air exchange rates as low as 0.1 to 0.3 per hour. In comparison with conventional homes, mobile homes and prefabricated housing units have lower mean air exchange rates and are of smaller volume. These characteristics make them particularly liable to indoor air pollution problems. In many new office buildings, construction techniques and ventilation practices also lead to low air exchange rates and the potential for air quality problems.

Although most time indoors is spent at home or at work, about 5% of each day is spent in transit (table 1). Transportation environments pose unique problems with regard to air quality. In most, air exchange rates are high, but the ratio of the number of occupants to air volume is much higher than in other environments. For example, commercial jets are designed to have several air exchanges per hour. In an automobile with the windows open or the ventilation fan operating, the number of air exchanges can range from 5 to 50 per hour. The occupant-to-volume ratios are much higher than found in almost every indoor environment used by the general public. Thus, substantial exposure to airborne pollutants may be sustained in transportation environments.

The ventilation systems of commercial aircraft are designed to pressurize the cabin, cool electronic equipment, vent lavatory and galley areas, and provide conditioned air for passenger and crew comfort (30). The air is drawn through compressors, conditioned by mixing with ambient air, and delivered through overhead systems. The air exchange on airplanes is usually quite high, although some aircraft, such as the new Boeing 767, are configured to recirculate 50% of the air. At full passenger load, only 7 cubic feet per minute (cfm) of fresh air per person would be supplied, only slightly greater than the minimum recommended in the current ventilation guidelines of the American Society of Heating, Refrigerating, and Air Conditioning Engineers.

Important and occasionally unique exposures to pollutants can be sustained in special environments, although little time may be spent in these locations on average. For example, sports arenas can be heavily contaminated with tobacco smoke (31), and motorized ice cleaning equipment can increase levels of CO and NO_2 in ice skating rinks (32). Exposure to volatile organic compounds (VOC) can take place in dry cleaning establishments and fabric stores (33). Camping lanterns and cooking stoves are potent sources of NO_2 and CO (34). In fact, fatalities have occurred to campers and explorers from CO poisoning (35, 36).

Sources of Indoor Air Pollution

Numerous sources of airborne contaminants have been identified in indoor environments (tables 2 and 3). The pollutant sources found in home, office, and transportation environments are listed in table 2. In table 3, we describe the sources of the principal pollutants and typical concentrations. Unvented combustion, evaporation of solvents, grinding, and abrasion can produce gaseous and particulate pollution indoors. Radon and its decay products accumulate indoors from soil, water, and building materials. Biological sources include growth of molds, fungi, and bacteria, and insects and pets.

In the home, the principal combustion sources are tobacco smoking, gas cooking stoves, and unvented kerosene heaters. Wood burning in stoves and fireplaces may also affect indoor air quality. Formaldehyde may be released from urea formaldehyde foam insulation (UFFI),

TABLE 3

THE PRINCIPAL INDOOR POLLUTANTS, THEIR SOURCES AND TYPICAL CONCENTRATIONS

Pollutant	Typical Sources	Pollutant Concentrations	Relevant Standards	Comments
Respirable particles	Tobacco smoke, unvented kerosene heaters, wood and coal stoves, fireplaces, outside air, attached facilities, occupant activities	> 500 μg/m³ bars, meetings, waiting rooms with smoking 100 to 500 μg/m³ typical for smoking sections of planes 10 to 100 μg/m³ typical of homes 1,000 μg/m³ with burning food or fireplaces	265 μg/m³ EPA 24-h standard ambient air 75 μg/m³ EPA annual standard ambient air 150 μg/m³ Japanese indoor standard	Current EPA standards are for total, and not only respirable suspended particles
NO, NO_2	Gas ranges and pilot lights, unvented kerosene and gas space heaters, gasoline engines, some gas floor furnaces, outside air	25 to 75 ppb typical range for homes with gas stoves 100 to 500 ppb peak values kitchens with gas stoves or kerosene gas heaters.	160 ppb 1-h maximum, WHO guideline 50 ppb annual average EPA ambient standard	No current EPA short-term standard
CO	Gas ranges and pilot lights, unvented kerosene and gas space heaters, tobacco smoke, back drafting of water heater or furnace or woodstove, gasoline engines, camping lanterns and stoves Attached garages, street level intake vents, hockey rinks	> 50 ppm when oven used for heating > 50 ppm attached garages, air intakes, arenas 2 to 15 ppm cooking with gas stove 2 to 10 ppm heavy smoking in homes, bars, and other locations	35 ppm EPA 1-h standard 9 ppm EPA 8-h standard	
CO_2	People, unvented kerosene and gas space heaters, tobacco smoke, outside air	320 to 400 ppm outdoor air 2,000 to 5,000 ppm crowded indoor environment, inadequate ventilation	1,000 ppm Japanese indoor air standard	CO_2 concentrations below 1,000 ppm usually indicate adequate fresh air supply for buildings.
Infectious, allergenic, irritating biological materials	Dust mites and cockroaches, animal dander, bacteria, fungi, viruses, pollens	Few systematic measurements of spores, bacteria, and viruses indoors Homes with mold problem, offices with water damage > 1,000 cfu/m³* Homes and offices without obvious problem: 500 ± 200 cfu/m³	None	Interpretations of a level depend on the specific agent; cfu/m is only an indicator
Formaldehyde	Urea formaldehyde foam insulation (UFFI), glues, fiberboard, pressed board, plywood, particle board, carpet backing and fabrics	0.1 to 0.8 ppm homes with UFFI 0.5 ppm average in mobile homes > 1 ppm in a few homes and mobile homes	0.2 to 0.5 ppm adopted by several states 0.1 ppm Sweden, new homes 0.7 ppm Sweden, maximum in old buildings 3 ppm U.S. OSHA 8-h time-weighted average	Formaldehyde concentrations in homes with UFFI decline by 50% every 2 to 3 yr.
Radon and radon daughters	Ground beneath a home, domestic water, and some utility natural gas	1.5 pCi/l estimated average in U.S. homes > 8 pCi/l in 3% to 5% homes	8 pCi/l NCRP action level 4 pCi/l EPA limit for uranium processing site homes 2 pCi/l ASHRAE guidelines 5 pCi/l Sweden, maximum, existing buildings 3 pCi/l Sweden, maximum, new buildings	Radon or radon daughters can be measured. Standards are for radon. Lung cancer risk results from radon daughters.
Volatile organic compounds: benzene, styrene, tetrachloroethylene; dichlorobenzene; methylene chloride; chloroform	Outgassing from water, plasticizers, solvents, paints, cleaning compounds, mothballs, resins, glues, gasoline, oils, combustion, art materials, photocopiers, personal care products	Typical indoor concentrations of selected compounds: benzene—15 μg/m³; 1,1,1 trichloroethane—20 μg/m³; chloroform—2 μg/m³; tetrachloroethylene—5 μg/m³; styrene—2 μg/m³, m,p-dichlorobenzene—4 μg/m³; m,p-xylene—15 μg/m³	No indoor standards for nonoccupational settings	EPA Carcinogenic Assessment Group potency factors available for many of the volatile organics.
Semivolatile organics: Chlorinated hydrocarbons, DDT, heptachlor, chlordane	Pesticides, transformer fluids, termicides, combustion of wood, tobacco, kerosene, and charcoal; wood preservatives, fungicides	Only limited data available	No indoor standards	
Semivolatile organics: polycyclic compounds, benzo(a)pyrene, polychlorinated biphenols	Herbicides, insecticides			
Asbestos	Insulation on building structural components, asbestos plaster around pipes and furnaces, tiles	No systematic measurements to determine typical fiber concentrations. > 1,000 ng/m³ when friable asbestos	2 fibers/cc OSHA 8-h time-weighted average	EPA and state attention has been on schools and office buildings. Domestic problems not evaluated

* cfu/m³ = colony-forming units/m³

from furnishings, and from household products. Volatile organic compounds have numerous sources within the home including tobacco smoking and household products. Radon, emitted by the earth under a home, can enter through cracks in the foundation, crawl spaces, sump holes, and other portals. Building materials, water, and utility natural gas may also be sources of radon.

In offices, tobacco smoking is an important source of respirable particulates. Formaldehyde and other VOC may be given off by building materials, furnishings, paints, waxes, supplies, and clean-

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ing solvents. Biological agents, which have proliferated on moist surfaces, may be dispersed by the office heating and cooling systems. Many of these same sources of air contamination are present in transportation environments.

Personal Exposure to Air Pollutants

Measurement of personal exposures to pollutants confirms the contributions of these indoor sources to total pollutant exposures (37). Direct personal monitoring has become possible with the development of passive sampling equipment and lightweight portable pump systems (38, 39). By combining personal sampling or fixed-location sampling with time-activity information, the relative contributions of various locations and sources to personal exposures can be estimated (37). Studies using these techniques have established the importance of indoor sources for exposure to respirable particulates, CO and NO_2.

For example, Spengler and associates (40) evaluated sources of variation in personal exposures to respirable particles among residents of 2 semirural communities in Tennessee. The ambient concentrations and personal exposures were uncorrelated, but the concentration of respirable particulates in the homes explained more than 60% of the variation in personal exposures. Reported tobacco smoke exposure alone accounted for less than 15% of the variability.

Quackenboss and colleagues (41) reported similar results from a study of personal NO_2 exposures of 35 adults and children living in the vicinity of an agricultural community in central Wisconsin. In this population more than 80% of the variance in week-long personal exposure to NO_2 was accounted for by variation in bedroom concentrations. In contrast, the studies of personal exposure have indicated the predominance of outdoor sources for some pollutants, e.g., O_3 (37).

Health Effects of Indoor Air Pollution

Introduction

We have described the sources of indoor air pollution and the principal pollutants that may be found in specific indoor environments. In this section, we address the health effects associated with these pollutants as well as information on the concentrations of the pollutants in indoor air. In discussing their health effects, we have broadly grouped the pollutants by their sources: combustion sources—tobacco smoke, NO_2, CO, and wood smoke; biological sources—infectious agents and allergens; and miscellaneous sources—radon and radon daughters, volatile organic compounds, and formaldehyde. The problem of building-related illnesses or "tight building syndrome," which cannot be linked to specific agents, is described separately. We do not review the hazards, primarily nonrespiratory, of exposure to pesticides. The National Research Council (42) has recently addressed the nonoccupational health risks of asbestiform fibers, and we do not cover this exposure. Finally, we do not consider the effects of pollutants generated by outdoor sources that penetrate indoors nor exposures in the work environment that are associated with well-recognized forms of occupational lung disease.

Tobacco Smoke

Introduction. Extensive toxicologic, experimental, and epidemiologic data, largely collected since the 1950s, have established that active cigarette smoking is a major preventable cause of morbidity and mortality (43). Involuntary exposure to tobacco smoke has only recently been investigated as a risk factor for disease in nonsmokers. Consequently, the evidence on involuntary smoking is more limited in scope than for active smoking, and controversy remains concerning certain associations of involuntary smoking with disease.

Nonsmokers inhale environmental tobacco smoke, the combination of the sidestream smoke that is released from the cigarette's burning end and the mainstream smoke exhaled by the active smoker (44). Comprehensive discussions of the chemistry of sidestream and of mainstream smoke are included in the 1979, 1984, and 1986 reports of the Surgeon General (43, 45, 46), in the 1981 report of the National Research Council on indoor air pollution (15), and in the 1986 report of the National Research Council on environmental tobacco smoke (47).

The exposures of involuntary and active smoking differ quantitatively and, to some extent, qualitatively (15, 45–48). Because of the lower temperature in the burning cone of the smoldering cigarette, most partial pyrolysis products are enriched in sidestream as compared to mainstream smoke. Consequently, sidestream smoke has higher concentrations of some toxic and carcinogenic substances than mainstream smoke; however, dilution by room air markedly reduces the concentrations inhaled by the involuntary smoker in comparison to those inhaled by the active smoker. Nevertheless, involuntary smoking is accompanied by exposure to many of the toxic agents generated by tobacco combustion (15, 45–48). The intake of tobacco smoke components by nonsmokers has been confirmed by studies using biological markers such as nicotine and its metabolite, cotinine. Thus, it is biologically plausible to hypothesize that exposure to environmental tobacco smoke is a risk factor for disease in nonsmokers. Active smokers must necessarily have greater exposure to environmental tobacco smoke than nonsmokers, but the consequences of smokers' active and passive exposures cannot be separately evaluated.

To date, research on passive smoking has focused on respiratory effects, although recent investigations have examined associations with diverse health effects including nonrespiratory cancers, ischemic heart disease, age at menopause (49), sudden infant death syndrome (50), and birth weight (51, 52). This review will emphasize the respiratory effects of involuntary smoking. Because the literature on passive smoking has been reviewed in this journal (53), in the 1984 and the 1986 reports of the Surgeon General (45, 46), and by the National Research Council (47), we will focus on the newer studies and the converging evidence for some effects of involuntary smoking. Symposia (18, 54, 55) and a monograph by Shephard (56) have also addressed the adverse health effects of involuntary smoking. Other reviews on selected aspects of the health effects of involuntary smoking have also been published (57–61).

Exposure to Environmental Tobacco Smoke. Tobacco smoke is a complex mixture of gases and particles that contains myriad chemical species (43, 45). Not surprisingly, tobacco smoking in indoor environments increases levels of respirable particulates, nicotine, polycyclic aromatic hydrocarbons, CO, acrolein, NO_2, and many other substances. The extent of the increase varies with the number of smokers, the intensity of their smoking, the ventilation rate of the indoor space, and the use of air cleaning devices. Several cigarette smoke components have been measured in indoor environments as markers of the contribution of tobacco combustion to indoor air pollution. Particulates have been measured most often; sidestream and mainstream smoke both

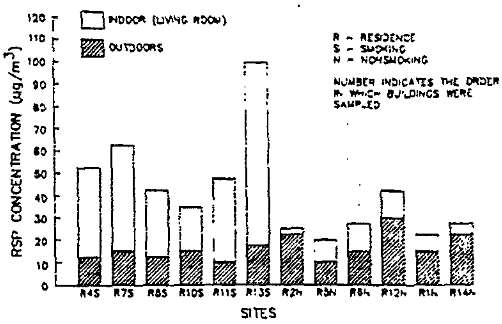
contain high concentrations of particles in the respirable size range (46, 47). However, surveys of indoor air quality based on measurement of total suspended particulate concentrations will not readily identify the excess mass indoors from environmental tobacco smoke. Studies of levels of environmental tobacco smoke components have been conducted largely in various public buildings; fewer studies have been conducted in the home and office environments (46, 47).

The contribution of smoking in the home to indoor air pollution has been demonstrated by studies involving personal monitoring and monitoring of homes for respirable particulates. Spengler and associates (62) monitored 80 homes for respirable particulate concentrations over several years and found that a smoker of 1 pack of cigarettes daily contributed about 20 μg/m³ to 24-h indoor particle concentrations. In homes with 2 or more heavy smokers, this study showed that the 24-h National Ambient Air Quality Standard of 260 μg/m³ for total suspended particulates could be exceeded. Because cigarettes are not smoked uniformly over the day, higher peak concentrations must occur when cigarettes are actually smoked. Therefore, short-term total suspended particulate concentrations of 500 to 1,000 μg/m³ are likely at the time when cigarettes are actually smoked. The dramatic effect of smoking in the home is shown in figure 2, which summarizes 24-h samples of respirable suspended particulates in residences (63). The variation in the excess indoor concentrations among residences was related to the number of smokers and the air exchange rates. Home 13 had an average air exchange rate of only 0.44 per hour.

Spengler and associates (40) measured the personal exposures to respirable particulates sustained by nonsmoking adults in 2 rural Tennessee communities. The mean 24-h exposures were substantially higher for those exposed to smoke at home.

Levels of other constituents of tobacco smoke have not been well characterized in homes. In the studies of Moschandreas and associates (63), only homes with smokers had iron, arsenic, and cadmium levels indoors that exceeded outdoor concentrations. Ambient cadmium ranged between 1 and 2.5 ng/m³ while the indoor home average concentrations ranged between 2 and 5 ng/m³ in the homes with heavy smoking. Under usual circumstances of smoking, the average emission rate of CO, 50 mg per cigarette, will not increase concentrations in a residence to the standards set for outdoor air (64).

Fig. 2. Average 24-h respirable suspended particulate (RSP) concentrations (μg/m³) outdoors and indoors in homes with and without smokers. (Redrawn with permission from reference 63.)



More extensive information is available on levels of environmental tobacco smoke in various public buildings. Monitoring in locations where smoking may be intense, such as bars and restaurants, has generally shown elevations of particulates and other markers of smoke pollution where smoking is taking place (46, 47). For example, Repace and Lowrey (65) used a piezobalance to sample aerosols in restaurants, bars, and other locations. They found that respirable particulate levels ranged up to 700 μg/m³ and varied with the intensity of smoking. Similar data have been reported for the office environment, though the information is more limited (46, 47).

Transportation environments may also be affected by cigarette smoking. Contamination of air in trains, buses, automobiles, airplanes, and submarines has been documented (46, 47). For example, a recent National Research Council Report (30) on air quality in airliners summarized studies of tobacco smoke pollutants in commercial aircraft. During a single flight, the NO_2 concentration varied with the number of passengers with a lighted cigarette. In another study, respirable particulates in the smoking section were measured at concentrations 5-fold or greater than in the nonsmoking section. Peaks as high as 1,000 μg/m³ were measured in the smoking section.

Markers of Exposure. Biological markers of tobacco smoke exposure have been used to describe the prevalence and the dosimetry of involuntary smoking. In both active and involuntary smokers, the detection of various smoke components or their metabolites in body fluids or alveolar air provides evidence of exposure, and levels of these markers can be used to gauge the intensity of exposure to tobacco smoke. The risks of involuntary smoking have also been estimated by comparing levels of biological markers in active and involuntary smokers.

At present, the most sensitive and specific markers for tobacco smoke exposure are nicotine and its metabolite, cotinine (47, 66). Neither nicotine or cotinine are present in body fluids without exposure to tobacco smoke. Because the circulating half-life of nicotine is generally less than 2 h (67), nicotine concentrations in body fluids reflect recent exposures. In contrast, cotinine has a half-life in the blood or plasma of active smokers that ranges from less than 10 h to about 40 h (68, 69). The half-life of cotinine tends to be longer in nonsmokers than in smokers (68). Hence, cotinine levels provide information about more chronic exposure to tobacco smoke in both active and involuntary smokers. Thiocyanate concentration in body fluids, concentration of CO in expired air, and carboxyhemoglobin level distinguish active smokers from nonsmokers, but these measures are not as accurate as cotinine for assessing involuntary exposure to tobacco smoke (66, 70, 71).

Recent reports described cotinine levels and their relationship to exposure in adult nonsmokers and in children (table 4). In adult nonsmokers, exposures at home, in the workplace, and in other settings determined cotinine concentrations in urine and saliva. The cotinine levels associated with involuntary smoking ranged from less than 1% to about 8% of cotinine levels measured in active smokers. As would be anticipated, smoking by parents was the predominant determinant of the cotinine levels in children. Greenberg and colleagues (75) found significantly higher concentrations of cotinine in the urine and saliva of infants exposed to cigarette smoke in their homes in comparison with unexposed controls. Urinary cotinine levels in the infants increased with the number of cig-

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TABLE 4

SELECTED STUDIES OF COTININE LEVELS IN NONSMOKERS

Population	Findings
100 adult patients attending clinics in London (72)	Cotinine levels parallel self-reported exposure. In nonsmoking, mean = 1.5 ng/ml in saliva; in active smokers, mean = 309.9 ng/ml.
151 adult males attending a clinic in London and 70 subjects from Oxford (73, 74)	Urinary cotinine level increased with reported duration of smoke exposure. In nonexposed nonsmokers, median level = 2.0 ng/ml; exposed nonsmokers, median = 6.0 ng/ml; active cigarette smokers, median = 1,645 ng/ml. Smoking by wife increased cotinine concentrations 3-fold in nonsmoking men.
51 infants attending clinics in North Carolina (76).	In nonexposed, median urinary level = 4 ng/mg creatinine; exposed, median = 351 ng/mg creatinine. Salivary creatinine levels higher in exposed.
472 nonsmoking adults in Japan (76).	Exposure at home and at work independently increased urinary cotinine level. In nonsmokers, median = 680 ng/mg creatinine; active smokers, median = 8,570 ng/mg creatinine.
569 schoolchildren, 11 to 16 yr of age, in Bristol, England (77).	Salivary cotinine level increased with the number of smoking parents. If neither parent smoked, median = 0.20 ng/ml; if both smoked, median = 2.70 ng/ml.
38 children, 3 to 15 months of age, attending a child center in North Carolina (78).	Serum cotinine level increased with the number of smokers in the home. In children without household exposure, mean = 1.0 ng/ml, exposed, mean = 4.1 ng/ml.
839 children and adults in a population sample in New Mexico (79)	Salivary cotinine level increased with the number of smokers in the home. If no smokers in the home, median level was zero. If 1 or more smokers, median was greater than zero and increased with the number of smokers.

arettes smoked during the previous 24 h by the mother. The findings were similar in another study of infants that was based on serum cotinine levels (78). Luck and Nau (80) have shown that cotinine and nicotine levels measured in infants breast-fed by smoking mothers reflect both the doses received from the breast milk and from involuntary smoking. In a study of schoolchildren in England, salivary cotinine levels rose with the number of smoking parents in the home (77). Similar data were recently reported from a large population-based sample in New Mexico (79).

The results of some investigations based on other markers of exposure have been similar. For example, Poulton and associates (81) reported that serum thiocyanate levels were significantly higher in children living with smokers. In fact, levels of thiocyanate are increased in umbilical cord blood if the mother lives with smokers (82). Nicotine levels in adults vary with report of recent exposure, and in several English studies all nonsmokers had measurable concentrations of nicotine in body fluids (66, 83, 84).

The results of these studies using biological markers have important implications for research on involuntary smoking. The studies provide ample evidence that involuntary exposure leads to absorption, circulation, and excretion of tobacco smoke components, and the studies confirm the high prevalence of involuntary smoking, as ascertained by questionnaire (85). The results further suggest that questionnaire methods for assessing recent exposure have some validity. These studies also demonstrate that saliva and urine samples can be readily obtained from large populations; thus, cotinine levels in body fluids could be used as a marker of exposure in large-scale epidemiologic research on involuntary smoking. However, further investigation is needed to define the relationship between inhaled nicotine and cotinine levels in body fluids, the extent to which cotinine levels index doses of other tobacco smoke components, and the range and determinants of cotinine half-life in nonsmokers. Further, a biological marker for cumulative exposure, which would facilitate investigation of chronic diseases, has not been identified.

Comparisons of levels of biological markers in smokers and nonsmokers have been made in order to estimate the relative intensities of active and involuntary smoking. However, a simple proportionality cannot be assumed between the ratio of the levels of markers in passive and active smokers and the relative doses of all tobacco smoke components. Nonetheless, several investigators have attempted to characterize involuntary smoking in terms of active smoking. For example, Foliart and coworkers (86) measured urinary excretion of nicotine in flight attendants during an 8-h flight and estimated that the average exposure was 0.12 to 0.25 mg of nicotine. Russell and colleagues (87) compared nicotine levels in nonsmokers exposed to tobacco smoke with levels achieved after infusion of known doses of nicotine. On the basis of this comparison, the investigators estimated that the average rate of nicotine absorption was 0.23 mg per hour in a smoky tavern, 0.36 mg per hour in an unventilated smoke-filled room, and 0.014 mg per hour from average daily exposure. In active smokers, the first cigarette of the day resulted in absorption of 1.4 mg of nicotine.

Nonmalignant Respiratory Effects. The 1983 "State of the Art" review on involuntary smoking (53) and the 1984 and the 1986 reports of the Surgeon General (45, 46) provide comprehensive summaries of the literature on respiratory effects of involuntary smoking other than lung cancer. These publications have concurred in concluding that for children passive smoking increases the occurrence of lower respiratory illness, particularly early in life, and increases the frequency of chronic respiratory symptoms. On the basis of primarily cross-sectional data, the 1984 report of the Surgeon General (45) also concluded that the children of smoking parents in comparison with those of nonsmokers had small reductions of lung function, but the long-term consequences of these changes were regarded as unknown. In the 2 yr between the 1984 and the 1986 reports, sufficient longitudinal evidence accumulated to support the conclusion in the 1986 report (46) that involuntary smoking reduces the rate of lung function growth during childhood. Only limited data pertaining to adults have been available, and definitive conclusions have not been made for adult populations.

The more recent data on children have generally supported the conclusions of the earlier review in this journal (53) and of the Surgeon General's reports. With regard to respiratory illness in infants, Pedreira and colleagues (88) prospectively monitored the incidence of lower respiratory illness in 1,144 infants followed in a pediatric practice. Office visits

for tracheitis and bronchitis were significantly more common for infants exposed to tobacco smoke at home. The effects of prenatal smoke exposure could not be separated from those of postnatal exposure in previous studies of lower respiratory illness. However, relevant data have been published from 2 populations. A prospective study in China of 1,058 infants of nonsmoking mothers demonstrated that paternal smoking increased the rate of hospitalization for respiratory illness during the first 18 months of life (89). A British cohort study suggested independent effects of prenatal and postnatal exposure on lower respiratory illness experience in early life (90).

Data from 2 large cross-sectional investigations demonstrated an association between parental smoking habits and lower respiratory illness before 2 yr of age (91, 92). Ware and associates (92) analyzed questionnaire information from 10,106 children, 6 to 9 yr of age at enrollment, who were participating in the Harvard Air Pollution Health Study in 6 U.S. cities. Smoking by both the mother and the father was associated with a higher frequency of reported physician-diagnosed respiratory illness before 2 yr of age. The relative odds for this illness variable increased progressively with the usual number of cigarettes smoked daily by the mother at the time of interview. In a prevalence survey of 1,355 Iowa children 6 to 12 yr of age, parental smoking significantly increased the risk of hospitalization for a chest illness before 2 yr of age (91). Although recall of past illnesses may be inaccurate (93), bias in reporting that depends upon parental smoking habits is unlikely.

Two recent studies did not show effects of involuntary smoking on respiratory illnesses in children. Gardner and colleagues (94) monitored 131 infants during the first year of life for viral infections by serology, cultures, and clinical examinations. Neither specific infections nor illnesses were associated with parental smoking habits. The study population was small, however, and did not have sufficient statistical power to examine effects in the range of interest. In a study based on data from a health maintenance organization, Vogt (95) found that household smoking characteristics did not influence use of outpatient care services for respiratory illness by children.

New studies have showed that children exposed to cigarette smoke in their homes are also at increased risk for middle ear disease. Both acute otitis media (96) and persistent middle ear effusions (97-99) have been associated with involuntary smoking.

The more recent studies continue to indicate increased respiratory symptoms in the children of smokers. In the Harvard Air Pollution Health Study, smoking by parents increased the frequency of cough and wheeze in their children by up to about 30% (92). Analysis of data from 3,482 nonsmoking children, collected in 1962 to 1965 in Tecumseh, Michigan, also indicated more frequent respiratory symptoms in the children of smokers (100, 101). Charlton (102) conducted a survey on cigarette smoking that included 15,709 English children 8 to 19 yr of age. In the nonsmoking children, the prevalence of frequent cough was significantly higher if either the father or the mother smoked, in comparison with the prevalence when neither parent smoked.

The findings of the newer studies are inconsistent on the relationship between passive smoking and wheezing and asthma. McConnochie and Roghmann (103) assessed predictors of wheeze in a retrospective cohort study of children who had mild bronchiolitis in infancy and of control children without illness. At a mean age of 8.3 yr, current exposure to tobacco smoke at home was a significant predictor of wheeze (odds ratio = 1.9, $p = 0.05$). Further analysis of data from the control children showed that maternal smoking significantly increased the prevalence of wheezing on follow-up in children from families with a history of respiratory allergy (104). In the study of children in Tecumseh, Michigan, parental smoking was associated with a higher prevalence of asthma at the initial examination and with a doubling of the risk for developing asthma during the 15-yr follow-up period (100, 101). Murray and Morrison (105) evaluated 94 asthmatic children 7 to 17 yr of age. Level of lung function, symptom frequency, and responsiveness to inhaled histamine were adversely affected by maternal smoking.

In contrast, Tashkin and associates (106) examined cross-sectional data from children 7 to 17 yr of age in the Los Angeles area and found no association between the smoking characteristics within the households and the prevalence of respiratory symptoms or asthma. In a prospective cohort study in New Zealand, parental smoking habits were not found to affect the incidence of asthma during the first 6 yr of life (107). In 1980, Weiss and associates (108) reported the results of a cross-sectional survey of respiratory symptoms in 650 children in Massachusetts. The prevalence of persistent wheeze, the most common symptom, increased significantly with the number of smoking parents but was unrelated to smoking by the children themselves. These investigators subsequently used cold air challenge to assess airways responsiveness in a sample of these children and found that airways reactivity was not related to maternal smoking history (109).

New studies have further documented the adverse effect of parental smoking on children's lung function, and longitudinal evidence on the consequences of passive smoking during lung growth and development was published. In the study of children in Tecumseh, Michigan, parental smoking was associated with reduced lung function, as assessed by spirometry (100, 101). The magnitude of effect varied with age, sex, and the index of exposure to parental smoking. In the survey in Los Angeles, maternal smoking was associated with average reductions of 3 to 8% for spirometric parameters in male subjects (106). The effects of paternal smoking were largest in boys less than 12 yr of age and were variable in girls. Ekwo and coworkers (91) found significantly greater response to inhaled bronchodilator in the children of cigarette smokers. In the Harvard Air Pollution Health Study, the FEV_1 of children whose mothers smoked at the time of spirometry was reduced by slightly less than 1% of predicted FEV_1 reduction (92). In contrast, Hosein and Corey (110) studied 1,357 children and did not find an effect of home exposure to tobacco smoke on FEV_1 level. Lebowitz and colleagues (111) also did not find effects of parental smoking, but only 271 children were included in the study population.

Based on cross-sectional data from children in East Boston, Massachusetts, Tager and associates (112) reported in 1979 that the level of FEF_{25-75} declined with the number of smoking parents in the household. In 1983, these investigators provided the results obtained with follow-up of these children over a 7-year period (113). Using a multivariate technique, Tager and associates showed that both maternal smoking and active smoking by the child reduced the growth rate of the FEV_1. The statistical model predicted effects of maternal smoking that are of a physiologically important magnitude. Lifelong exposure of a child to a smoking mother was estimated to reduce growth of the FEV_1 by 10.7, 9.5,

and 7.0% after 1, 2, and 5 yr of follow-up, respectively.

Recent longitudinal data from the Harvard Air Pollution Health Study also showed reduced growth of the FEV_1 in children whose mothers smoked cigarettes (114). The growth rate of the FEV_1 from ages 6 through 10 yr was calculated for 7,834 white children. Although all representations of exposure to parental smoking were associated with reduced growth rate of the FEV_1, only the level of maternal smoking in packs per day attained statistical significance at $p < 0.05$. From ages 6 through 10 yr, the statistical model estimated that FEV_1 growth rate is reduced by 0.17% per pack of cigarettes smoked daily by the mother. This effect was somewhat smaller than that reported earlier by Tager and associates (113), although if extrapolated to age 20 yr, a cumulative effect of 2.8% is predicted. The 2 sets of data were also analyzed with noncomparable techniques, and the study populations may have differing levels of exposure to passive smoking.

Burchfiel (100, 101) examined the effects of parental smoking on 15-yr lung function change of subjects in the Tecumseh study, first examined at ages 10 through 19 yr. In the female subjects who remained nonsmokers across the follow-up period, parental smoking was not associated with lung function change. In nonsmoking males, parental smoking reduced the growth of the FEV_1, FVC, and $\dot{V}max_{50}$, although the sample size was limited and the effects were not statistically significant. For the FEV_1 in males, the analysis estimated 7.4% and 9.4% reductions in 15-yr growth associated with 1 or 2 smoking parents, respectively.

Some new information has become available for adults since the previous reviews, which cited data from only 4 epidemiologic studies. The ratio of hydroxyproline to creatinine in urine was used by Japanese investigators as a marker of lung injury (115). In women passively exposed to cigarette smoke, this ratio increased with the extent of daily exposure. However, in a study in Germany, the hydroxyproline to creatinine ratio in nonsmokers did not vary with passive smoke exposure (116). Moreover, Read and Thornton (117) reported that in experimental studies with rats, the hydroxyproline to creatinine ratio actually decreased with increasing exposure to smoke. They also reported that in humans both hydroxyproline and creatinine individually increased with increased nicotine absorption from active smoking in males but not in females (117). The ratio of the two, however, was not associated with increased nicotine excretion in either sex.

The results of several of the more recent epidemiologic studies indicate possible chronic effects of passive smoking on lung function in adults. The results of an investigation of 163 nonsmoking women in the Netherlands suggested adverse effects of tobacco smoke exposure in the home (118, 119). Cross-sectional analysis of spirometric data collected in 1982 showed reductions of most parameters in association with tobacco smoke exposure in the home, although the effect was significant only for flows at higher lung volumes. In a sample of the women, domestic tobacco smoke exposure was not associated with longitudinal decline of lung function during the period 1965 to 1982. In baseline data for a cohort study in Scotland, respiratory symptoms tended to be more prevalent in nonsmokers living with smokers in comparison to nonsmokers living with nonsmokers (120).

Other studies have not indicated chronic effects of passive tobacco smoke exposure on adult nonsmokers. Jones and associates (121) conducted a case-control study of 20- to 39-yr-old nonsmoking women in the Tecumseh Community Health Study cohort. Subjects from the highest and lowest quartiles of the lung function distribution had comparable exposure to smokers in the home. Kentner and colleagues (122) in a study conducted in Germany examined the effects of passive and active smoking in 1,351 white collar workers. Self-reported exposure to environmental tobacco smoke at home and at work was not associated with reduction of lung function, as assessed by spirometry. In a small case-control study, marriage to a smoker was not associated with excess risk for chronic bronchitis (123).

New experimental and epidemiologic studies have not consistently shown acute effects of passive smoking on lung function level in asthmatic and nonasthmatic children and adults. As described above, Murray and Morrison (105) found lower ventilatory function in asthmatic children with smoking mothers. In a population sample in Tucson, Arizona, Lebowitz (124, 125) examined the relationship between passive smoking and daily symptom occurrence and daily level of peak flow. Statistically significant effects of tobacco smoke exposure were not found for either outcome in the 229 children and adults. In an experimental study, 1-h chamber exposure of young asthmatics to cigarette smoke did not reduce expiratory flow rates and was, in fact, followed by a small decrease in nonspecific airways reactivity (126).

The accumulating evidence since previous reviews continues to demonstrate adverse effects of passive smoking on the lungs of children. Data from large populations showed significant effects on lung function level and symptom occurrence (91, 92, 102, 106). Results from follow-up of the East Boston, the Harvard, and the Tecumseh study cohorts (100, 101, 113, 114) suggested that the effects on lung function should not be dismissed as clinically insignificant.

Important research questions pertaining to passive smoking and the child's lung remain unanswered, however (46, 127). The mechanisms of injury have not been established, and the relative importance of exposures *in utero*, during infancy, and later in childhood has not been examined. Nevertheless, the available evidence of adverse effects does provide sufficient rationale for intervention. In contrast to the evidence for children, the data on adults are more variable and do not yet permit conclusive statements concerning passive smoking during adulthood and reductions of lung function and increased respiratory symptom occurrence.

Lung Cancer. In 1981, reports were published from Japan (128) and from Greece (129) that indicated increased lung cancer risk in nonsmoking women married to cigarette smokers. Subsequently, this controversial association has been examined in investigations conducted in the United States, Scotland, Japan, and Hong Kong. The association of involuntary smoking with lung cancer derives biological plausibility from the presence of carcinogens in sidestream smoke and the lack of a documented threshold dose for respiratory carcinogenesis in active smokers (130). Further, mutagenic activity can be found in the urine of nonsmokers after passive exposure to tobacco smoke (131, 132).

Time trends of lung cancer mortality in nonsmokers have been examined with the rationale that temporally increasing exposure to environmental tobacco smoke should be paralleled by increasing mortality rates. Enstrom (133) calculated nationwide lung cancer mortality rates for 1914 to 1968 and concluded that a real increase had occurred among nonsmoking males after 1935. However, occupational and environmental exposures

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TABLE 5

COHORT AND CASE-CONTROL STUDIES OF PASSIVE EXPOSURE TO TOBACCO SMOKE AND LUNG CANCER

Study	Findings	Comment
Prospective cohort study in Japan of 91,540 nonsmoking females, 1966–1981 (136).	Age-occupation adjusted SMR,* by husband smoking: Nonsmokers—1.00 Ex-smokers—1.36 < 20/day—1.45 ≥ 20/day—1.91	Trend statistically significant. All histologies.
Case-control study in Greece of 40 nonsmoking female cases, 149 controls, 1978–1980 (129)	Odds ratios by husband smoking: Nonsmokers—1.0 Ex-smokers—1.8 Current smokers ≤ 20/day—2.4 > 20/day—3.4	Trend statistically significant. Histologies other than adenocarcinoma and bronchioloalveolar carcinoma.
Prospective cohort study in the U.S. of 176,139 nonsmoking females, 1960–1972 (134).	Age-adjusted SMR, by husband smoking: Nonsmokers—1.00 Current smokers < 20/day—1.27 ≥ 20/day—1.10	All histologies. Effect of husband smoking not significant
Case-control study in Hong Kong of 84 female cases and 139 controls, 1976–1977 (137, 138).	Crude odds ratio of 0.75 associated with smoking spouse	All histologies. Two reports are inconsistent on the exposure variable.
Case-control study in the U.S with 22 female and 8 male nonsmoking cases. 133 female and 180 male controls (139).	Odds ratios by spouse smoking: Nonsmokers—1.00 < 40 pack years—1.48 ≥ 41 pack years—3.11	Significant increase for > 41 pack years. Bronchioloalveolar carcinoma excluded.
Case-control study in the USA. 25 male and 53 female nonsmoking cases with matched controls, 1971–1980 (140).	Odds ratio not significantly increased for current exposure at home: Males—1.26 Females—0.92	All histologies Findings negative for spouse smoking variable as well.
Prospective cohort study in Scotland of 8,128 males and females, 1972–1982 (120).	Age-adjusted mortality ratios for domestic exposure: Males—3.25 Females—1.00	Preliminary, small numbers of cases.
Case-control study in Hong Kong with 88 nonsmoking female cases, 1981–1982 (141, 142).	Odds ratio of 1.24 (p > 0.40) for combined home and workplace exposure. No association with cumulative hours of exposure.	All histologies
Case-control study in the U.S. with 31 nonsmoking and 189 smoking female cases (143)	No significant effects of exposure from parents, spouse, or workplace in smokers and nonsmokers.	Adenocarcinoma and squamous cell carcinoma only.
Case-control study in the U.S. with 134 nonsmoking female cases (135)	Nonsignificant odds ratio of 1.22 if husband smoked. Significantly increased odds ratio of 2.11 if husband smoked 20 or more cigarettes daily at home. Significant trend with number of cigarettes smoked at home by the husband.	All histologies Careful exclusion of smokers from the case group.
Case-control study in England with 15 male and 32 female nonsmoking cases, and 30 male and 66 female nonsmoking controls (123).	Overall odds ratio for spouse smoking of 1.1.	Hospital-based study.
Case-control study in Japan with 19 male and 94 female nonsmoking cases, and 110 male and 270 female nonsmoking controls (144).	For females, odds ratio of 1.5 if husband smoked, for males, odds ratio of 1.8 if wife smoked.	Clinical or radiologic diagnosis for 43%. All histologies.
Case-control study in Louisiana, Texas, and New Jersey with 99 nonsmoking cases and 736 controls (145).	Adjusted odds ratio for marriage to a smoking spouse was 1.5.	Nearly 100% histologic confirmation All histologies.
Case-control study in New Mexico with 28 nonsmoking cases and 292 nonsmoking controls (146).	Adjusted odds ratio for marriage to a smoking spouse was 3.2. No effect in active smokers.	All histologies other than bronchioloalveolar carcinoma.

* Standardized mortality ratio

other than environmental tobacco smoke could explain the apparent mortality rate increase in males. Garfinkel (134) did not identify similar trends in nonsmokers in the Dorn study of male U.S. veterans, 1954 to 1969, nor in the American Cancer Society's study of males and females, 1960 to 1972.

Epidemiologists have tested the association between lung cancer and involuntary smoking using conventional designs: the case-control and cohort studies. The results of both study designs may be affected by inaccurate assessment of exposure to environmental tobacco smoke, by inadequate information on personal smoking habits that leads to classification of smokers as nonsmokers, and by the misdiagnosis of a cancer at another site as primary cancer of the lung. For example, in the case-control study reported by Garfinkel and colleagues (135), 13% of cases originally diagnosed as lung cancer were reclassified to other sites after histological review and 40% of the cases initially classified as nonsmokers by chart review were found to be smokers on interview. The difficulty of accurately estimating exposures with questionnaires and descriptions of a spouse's smoking may partly explain the variable findings of the published studies. In fact, the validity and reliability of questionnaires on involuntary smoke exposure have yet to be comprehensively evaluated.

The evidence from the case-control and the cohort studies does not uniformly indicate increased lung cancer risk in persons exposed to environmental tobacco smoke, but most of the studies indicate increased risk in nonsmokers married to smokers (table 5). Hirayama

(128) conducted a prospective cohort study of 91,540 nonsmoking women in Japan. Standardized mortality ratios for lung cancer increased significantly with the amount smoked by the husbands. The findings could not be explained by confounding factors and were unchanged when follow-up of the study group was extended (136). After its publication, this article received intensive scrutiny, and correspondence in the *British Medical Journal* raised concerns about statistical methodology, population selection, uncontrolled confounding, and the seemingly high relative risk; in his responses, Hirayama satisfactorily rebuffed most of these criticisms, although he could not eliminate the possibility of unreported smoking by women classified as nonsmokers (147). Based on the same cohort, Hirayama has also reported significantly elevated standardized mortality ratios for lung cancer of 2.1 and 2.3 in nonsmoking men with wives smoking 1 to 19 cigarettes and 20 or more cigarettes daily, respectively (136).

In 1981, Trichopoulos and colleagues (129) also reported increased lung cancer risk in nonsmoking women married to cigarette smokers. These investigators conducted a case-control study in Athens, Greece, that included cases with a diagnosis other than adenocarcinoma or bronchioalveolar carcinoma and controls selected at a hospital for orthopedic disorders. The findings were unchanged with expansion of the study population (148).

The results of other subsequently reported case-control studies have also demonstrated statistically significant associations between involuntary smoking and lung cancer (135, 144–146) (table 5). The findings from the more recent reports greatly strengthen the evidence from the earlier studies. Several of the newer studies included relatively large numbers of nonsmokers (135, 144, 145). Furthermore, in most of these studies, involuntary smoking was assessed in greater detail than in the earlier reports so that exposure-response relationships could be more fully examined.

The results of 2 other investigations have also been interpreted as showing an increased lung cancer risk associated with passive smoking, although both have methodologic limitations. In Germany, Knoth and colleagues (149) described a series of 59 female lung cancer cases of which 39 were in nonsmokers. Based on census data, the report by Knoth and colleagues projected that a much greater than expected proportion of these nonsmokers had lived in households with smokers. This report did not include an appropriate comparison series, however, and the suitability of substituting census data was not addressed by the authors. In another recent report, Gillis and associates (120) described the preliminary results of a cohort study of 16,171 males and females in western Scotland; domestic exposure to tobacco smoke increased the lung cancer risk for nonsmoking men but not for women. The report was based on only 16 cases of lung cancer in nonsmokers, however.

The results of other investigations indicate lesser or no effects of exposure to environmental tobacco smoke (table 5). In these studies, however, confidence limits for the relative risks associated with marriage to a smoker are wide and overlap with the confidence limits in the studies with significant results (47). Two separate case-control studies in Hong Kong, where lung cancer incidence rates in females are particularly high, did not indicate excess risk from passive smoking (137, 138, 141, 142). In the more recent of the 2 studies, the questionnaire comprehensively assessed cumulative exposure from home and workplace sources (141, 142). Lee and colleagues (123) reported a hospital-based case-control study in England. Although the investigators considered that their findings indicated little or no effect of involuntary smoking, the case series was small.

The results of the American Cancer Society's prospective cohort study of mortality in 176,139 nonsmoking women have also been construed by many as negative (134). However, the standardized mortality ratios for the nonsmoking women with husbands who smoked were greater than unity but not significantly greater. Repace (150) has suggested that the mortality ratios in the American Cancer Society cohort have been reduced by misclassification introduced by workplace exposures, a factor not considered in the original analyses. Recent and preliminary results from a nationwide case-control study also did not demonstrate increased lung cancer risk from domestic exposure to tobacco smoke (140). In another case-control study that was performed in Los Angeles, Wu and colleagues (143) did not find significantly increased risk for adenocarcinoma associated with involuntary smoking in smoking and nonsmoking women. These investigators estimated exposure from parental smoking, spouse smoking, and workplace sources. The relative risk for lung cancer was slightly, but not significantly, increased by exposure from spouse smoking and from smoking by coworkers.

At present, relatively few investigations provide data on the hypothesis that involuntary smoking is a risk factor for lung cancer. The extent of data contrasts with the more extensive literature cited in the 1964 Surgeon General's Report, which characterized active cigarette smoking as a cause of lung cancer (151). The variability of the data on involuntary smoking also contrasts with that on active smoking. However, most of the studies on involuntary smoking and lung cancer have small numbers of cases, and confidence intervals for the effect of involuntary smoking in the various studies would overlap. Variation in the results of the studies may also reflect random and nonrandom errors in the classification of exposure to environmental tobacco smoke. In fact, the problems of dose estimation seem more difficult for lung cancer than for other health effects of involuntary smoking. The relevant exposures may begin at birth and occur under a wide variety of circumstances. Thus, some inconsistency of the studies would be anticipated.

In spite of the variable epidemiologic evidence, environmental tobacco smoke has been recently characterized as a respiratory carcinogen. The International Agency for Research on Cancer of the World Health Organization (152) has concluded that "passive smoking gives rise to some risk of cancer." The agency supported this conclusion in its monograph on tobacco smoking by citing the characteristics of sidestream and mainstream smoke, the absorption of tobacco smoke materials during involuntary smoking, and the nature of dose-response relationships for carcinogenesis. Appropriately, the International Agency for Research on Cancer argued on the basis of biological plausibility rather than on the basis of epidemiologic evidence.

The National Research Council (47) and the U.S. Surgeon General (46) have also concluded that involuntary smoking increases the incidence of lung cancer in nonsmokers. In reaching this conclusion, the National Research Council (47) cited the biological plausibility of an association between environmental tobacco smoke exposure and lung cancer and the supporting epidemiologic evidence. This report carefully considered the sources of bias that may have affected the epidemiologic studies. Based on a

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pooled analysis of the epidemiologic data and adjustment for bias, the report's authors concluded that the best estimate for the excess risk of lung cancer in nonsmokers married to smokers was 25%. The 1986 report of the Surgeon General (46) characterized involuntary smoking as a cause of lung cancer in nonsmokers. This conclusion was based on the extensive information already available on the carcinogenicity of active smoking, on the qualitative similarities between environmental tobacco smoke and mainstream smoke, and on the epidemiologic data on involuntary smoking.

The extent of the lung cancer hazard associated with involuntary smoking in the United States remains uncertain, however (46, 153). The epidemiologic studies provide varying and imprecise measures of risk, and dosimetric aspects of involuntary smoking in the respiratory tract are not yet well understood. Risk estimation procedures have been used to describe the lung cancer risk associated with involuntary smoking, but assumptions and simplifications must be made in order to use this method. For example, Repace and Lowrey (154) have recently calculated that approximately 5,000 lung cancer deaths occur annually in U.S. nonsmokers as a result of involuntary smoking. The results of more refined risk estimation models should be forthcoming.

Other Cancers. Recent reports suggest that environmental tobacco smoke exposure may increase risk of cancer at sites other than the lung. One study found that in children, maternal exposure to environmental tobacco smoke during pregnancy was associated with increased risk of brain tumors (155), and in another study paternal but not maternal smoking increased the risk of childhood rhabdomyosarcoma (156). Such effects might arise from smoking-induced changes in germ cells of the parents or through transplacental exposure rather than as a direct effect of smoke inhalation (157, 158).

In adults, involuntary smoking was linked to a generally increased risk of malignancy and to excess risk at specific sites. Miller (159) interviewed surviving relatives of 537 deceased nonsmoking women in western Pennsylvania concerning the smoking habits of their husbands. A significantly increased risk of cancer death (odds ratio = 1.94, $p < 0.05$) was found in women who were married to smokers and were also not employed outside their homes. The large number of potential subjects who were not interviewed and the possibility of information bias detract from this report.

Sandler and colleagues (160–162) conducted a case-control study on the effects of childhood and adulthood exposures to environmental tobacco smoke on the risk of cancer. The 518 cases included all cancers other than basal cell cancer of the skin; the cases and the matched controls were between 15 and 59 yr of age. For all sites combined, significantly increased risk was found for parental smoking (crude odds ratio = 1.6) and for marriage to a smoking spouse (crude odds ratio = 1.5); the effects of these 2 exposures were independent (162). Significant associations were also found for some individual sites: for childhood exposure (161), maternal and paternal smoking increased the risk of hematopoietic malignancy, and for adulthood exposure (160), spouse's smoking increased the risk for cancers of the female breast, female genital system, and the endocrine system. These findings are primarily hypothesis generating and require replication. In a case-control study, such as reported by Sandler and colleagues, biased information on exposure to environmental tobacco smoke is of particular concern. Hirayama (136) has reported significantly increased mortality from nasal sinus cancers and from brain tumors in nonsmoking women married to smokers in the Japanese cohort. In a case-control study of bladder cancer, involuntary smoke exposure at home and at work did not increase risk (163).

These associations of involuntary smoking with cancer at diverse nonrespiratory sites cannot be readily supported with arguments for biological plausibility. Increased risks at some of the sites, e.g, cancer of the nasal sinus and female breast cancer, have not been observed in active smokers (130). In fact, the International Agency for Research on Cancer has concluded that effects would not be produced in passive smokers that would not be produced to a larger extent in active smokers (152).

Cardiovascular Disease. While extensive data establish active cigarette smoking as a causal risk factor for cardiovascular diseases (164), only a few studies have addressed involuntary smoking as a risk factor for these diseases. In the cohort of nonsmoking Japanese women, Hirayama (165) found a small but statistically significant increased risk of death from ischemic heart disease associated with the husband's smoking. Garland and associates (166) prospectively determined mortality from ischemic heart disease in nonsmoking older women residing in southern California. After adjustment for established risk factors, marriage to a smoking spouse was associated with a relative risk of 2.7 ($p < 0.10$). Gillis and colleagues (120) assessed the baseline prevalence of cardiovascular symptoms and major electrocardiographic abnormalities in a population sample residing in Scotland and then determined cause-specific mortality for up to 10 yr of follow-up. In their preliminary report, involuntary smoking was not associated with the prevalence of cardiovascular symptoms at baseline nor with cardiovascular mortality on follow-up. A case-control study in England did not show increased risk for ischemic heart disease or for stroke in nonsmokers married to smokers (123).

Total Mortality. Two cohort studies, the previously mentioned study in Scotland (120) and an investigation of civil servants and their spouses in Amsterdam (167), provided information on involuntary smoking and mortality from all causes. In the Scottish study, total mortality was increased for women living with a smoker but not for men (120). In contrast, mortality was not increased for nonsmoking female subjects in the study in Amsterdam (167). Neither study directly controlled for other factors that influence total mortality.

Summary. The effects of active smoking and the toxicology of cigarette smoke have been comprehensively examined. When considered in the context of that extensive information, the evidence on involuntary smoking supports conclusions concerning certain health effects. In children, involuntary smoking increases the occurrence of lower respiratory illness during infancy, increases the frequency of chronic respiratory symptoms, and reduces the level of lung function. In adults, involuntary smoking is a cause of lung cancer, but associations with other diseases have not been unequivocally established.

Nitrogen Dioxide

Introduction. Nitrogen dioxide causes lung damage at high concentrations (168, 169), but effects at levels currently encountered in outdoor and indoor air have been difficult to characterize. Early studies focused on the health effects of ambient NO_2 (25). However, in the late 1970s, investigators recognized that indoor NO_2 sources were also contributing to personal exposure and that indoor concentrations often exceeded outdoor concentrations in many homes (15). Consequently, more recent studies have em-

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TABLE 6

EFFECTS OF GAS COOKING ON RESPIRATORY ILLNESSES AND SYMPTOMS IN CHILDREN

Study Population	Outcome Measure	Results
British Studies:		
5,758 children, 6 to 11 yr of age, England and Scotland (178).	Major respiratory symptoms and diseases individually and as a single composite variable describing the presence of any 1 of 6 symptoms or diseases.	Significant asociations with gas cooking of selected symptoms and diseases, and of a composite variable.
2,408 children, 42% of original 5,758 in above study (179).	Single composite variable as described above.	Relative risk for composite variable generally exceeded 1.0; risk varied and decreased with age.
4,827 children, 5 to 11 yr of age, England and Scotland (179).	Single composite variable as described above.	Significant effect of gas stoves on composite variable in urban areas only
808 children, 6 to 7 yr of age, United Kingdom (180).	Single composite variable as described above.	Borderline significant association between composite variable and gas stoves. Increased prevalence as bedroom NO_2 levels increased in a sample with measurements ($n = 80$).
191 children, 5 to 6 yr of age, England (181).	Single composite variable as described above.	No significant association between bedroom NO_2 levels and prevalence of composite variable.
390 infants, 0 to 1 yr of age, England (182).	Respiratory illnesses and symptoms requiring physician visits, assessed prospectively.	No association between gas stove use and respiratory illnesses and symptoms.
1,565 infants, 0 to 1 yr of age, England (183).	Respiratory illnesses and hospitalizations assessed prospectively to 1 yr.	No significant association between illness or hospitalizations and use of gas for cooking
Ohio Studies		
441 upper-middle class families including 898 children less than 16 yr of age (184).	Incidence of acute respiratory illness, determined by bi-weekly telephone calls.	Respiratory illness incidence similar in homes using gas and electric stoves.
120 families from first study, including 176 children less than 12 yr of age (185)	Incidence of acute respiratory illness, determined by bi-weekly telephone calls and validated by home visits.	Respiratory illness incidence similar in homes using gas and electric stoves.
Harvard Air Pollution Health Study:		
8,120 children, 6 to 10 yr of age, 6 U.S cities (186, 187).	History of MD diagnosed bronchitis, of serious respiratory illness before age 2, of respiratory illness in last year	Significant association between current use of gas stove and history of respiratory illness before age 2 (odds ratio = 1.23)
10,106 children, 6 to 10 yr of age, 6 U.S cities Expansion of above study (92).	Same as above.	Odds ratio for history of respiratory illness before age 2 decreased to 1.12 ($p = 0.07$).
Other Studies:		
676 children, 3rd and 4th grades, Arizona (188).	Prevalence of asthma, wheeze, sputum, cough as determined by parent-completed questionnaire.	Significant association between use of gas stove and prevalence of cough (prevalence rate ratio = 1.97).
4,071 children, 5 to 14 yr of age, Pennsylvania (189).	Major respiratory illnesses and symptoms as determined by parent-completed questionnaire.	No significant association between use of gas stove and any symptom or illness variable
1,138 children, 6 to 12 yr of age, Iowa (91).	Major respiratory symptoms and illnesses as determined by parent-completed questionnaire	Significant association between current gas stove use and hospitalization for respiratory illness before age 2 (odds ratio = 2.4)
121 children, 0 to 13 yr of age, Connecticut (190)	Number of days of illness.	Number of days of illness associated with average hours of heater use
231 children, 6 yr of age, Netherlands (191).	Comparison of NO_2 levels in homes of cases (children with asthma) and controls	NO_2 distributions similar in homes of cases and controls

phasized sources and effects of indoor NO_2 concentrations.

Exposure. Combustion of gas during cooking and the burning of pilot lights releases nitric oxide (NO), NO_2, CO, CO_2, and water. On average, normal use of an unvented gas cooking range adds 25 parts per billion (ppb) of NO_2 to the background concentration in a home (170). The increase is greater during cold weather when the air exchange rate is usually reduced. During cooking with a gas range, peak levels in the kitchen may reach 200 to 400 ppb (10). Therefore, measured personal exposures to NO_2 are higher for persons living in homes with gas stoves than for persons living in homes with electric stoves (26, 41, 170).

Exposure to NO_2 from gas cooking stoves and ovens is widespread. About 50% of homes in the United States have gas cooking appliances; in some urban areas, such as Los Angeles, more than 90% of homes are equipped with gas appliances (171). The potential importance of NO_2 exposure indoors for health is underscored by comparison of the federal standard set for ambient air, 50 ppb annual average, with levels measured in homes with gas cooking appliances. Sexton and associates (172) used data generated by personal, indoor, and outdoor monitoring to develop a computer model for personal and indoor exposure. The model was applied to residents of 6 U.S. cities. Although none of the cities experienced concentrations above the federal standard in outdoor air, the model predicted that more than 25% of the residents of homes with gas ranges would have annual personal exposures over 50 ppb if ambient NO_2 concentrations averaged 30 ppb.

Health Effects. Most studies of the relationship between residential exposure to NO_2 and health have focused on respiratory symptoms and illnesses and on level of pulmonary function. Experimen-

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TABLE 7

EFFECTS OF GAS COOKING ON LUNG FUNCTION IN CHILDREN

Study Population	Lung Function Measure	Results
808 children, 6 to 7 yr of age, United Kingdom (180).	PEFR, $FEV_{0.75}$, FEF_{25-75}	No association with NO_2 levels or presence of gas stove.
898 children, 0 to 15 yr of age, from 441 families, Ohio (184).	FVC, $FEV_{0.75}$	Data on children not presented separately. No association with presence of a gas stove.
8,120 children, 6 to 10 yr of age, 6 U.S. cities (186, 187).	FVC, FEV_1	Overall reduction of 16 ml and 18 ml, respectively, for FEV_1 and FVC in children from homes with gas stoves.
16,689 children, 6 to 13 yr of age, 7 areas in U.S. (192).	$FEV_{0.75}$	Significant reduction of 19 ml associated with gas stove use in older girls only.
676 children, 3rd and 4th graders, Arizona (188)	FEV_1	No effect of gas stoves on pulmonary level or rate of growth.
183 children, 6 to 12 yr of age, Iowa (91).	FEV_1, FEF_{75}, FEF_{25-75}	No change after isoproterenol challenge in children from homes with gas stoves.
9,720 children, 6 to 10 yr of age, 6 U.S. cities (92).	FEV_1, FVC	Significant reduction in FEV_1, of 0.6% and FVC of 0.7%. Not significant after adjustment for parental education.
3,175 children, 5 to 14 yr of age, Pennsylvania (193).	FVC, $FEV_{0.75}$, FEF_{25-75}, $Vmax_{75}$, $Vmax_{90}$	No association with use of gas stove

tal investigations support the choice of these outcome measures; NO_2 may damage the lung directly through its oxidant properties or indirectly by increasing susceptibility to respiratory infections (169, 173). In animal models, NO_2 reduces the efficacy of specific lung defense mechanisms, and effects on mucociliary clearance, the alveolar macrophage, and the immune system have been demonstrated (169, 174, 175).

Data on the health effects of NO_2 concentrations likely to be encountered by the general population are derived from experimental and epidemiologic studies. The results of some human exposure studies imply that levels comparable to those measured in homes may increase airways reactivity in some asthmatics, but the results of other studies are inconsistent (175–177). Although experimental studies are useful for describing effects of controlled exposures, they cannot address the issue of chronic effects from chronic lower level exposures. Numerous epidemiologic investigations have now been carried out to assess their relationship.

The majority of these investigations were cross-sectional surveys of schoolchildren (tables 6 and 7). The investigators generally assessed current symptom status and retrospective illness histories, as obtained by parent-completed questionnaire, and pulmonary function. Although NO_2 levels were measured in several of the investigations (180, 181, 194), exposure was most often assessed by simple questions concerning type of fuel used for cooking. Consistent evidence of excess respiratory symptoms and illnesses in children exposed to gas stoves has not been demonstrated (table 6).

Early reports from two cross-sectional surveys of schoolchildren in Great Britain indicated that children from homes with gas stoves had a higher prevalence of respiratory symptoms than children from homes with electric stoves (178, 179). When one of the survey groups was followed longitudinally, however, the relative risks associated with gas stove use became highly variable and tended to decrease as the children grew older (179). These same British investigators surveyed a third group of 808 schoolchildren, and measured NO_2 concentrations in the homes of a small sample (n = 80 or 103). The prevalence of respiratory symptoms was higher in children from homes where gas was used for cooking and increased with higher bedroom NO_2 concentrations, although both effects were of borderline statistical significance (180). A similar association between measured NO_2 and respiratory symptoms was not replicated, however, when these same investigators subsequently studied another sample of 183 children (181). Two prospective studies of infants in Great Britain also failed to demonstrate an association between the use of gas for cooking and respiratory illness (182, 183).

Data on children from the United States are similarly inconsistent. Two large cross-sectional studies, one involving the Harvard Air Pollution Health Study (186, 187) and the other involving schoolchildren in Iowa (91), have demonstrated that reports of serious respiratory illness before 2 yr of age (186, 187) and hospitalization for respiratory illness before 2 yr of age (91) were more common among children from homes with gas stoves. When the original cohort in the Harvard Air Pollution Health Study was expanded, however, the odds ratio of 1.23 for serious respiratory illness before 2 yr of age decreased to 1.12 ($p = 0.07$). In the study of Ekwo and associates (91), the effect of exposure to a gas stove varied strongly and inconsistently with parental smoking habits. The effect was absent in homes where 1 parent smoked, largest where both parents smoked, and intermediate where neither smoked. This pattern of interaction cannot be readily interpreted biologically. Schenker and colleagues (189) found no association between type of cooking stove and current respiratory symptoms or previous illness history in a cross-sectional survey of 4,071 schoolchildren in western Pennsylvania.

The relationship between stove type and respiratory illness has also been studied prospectively. Keller and colleagues (184, 185), in a study of 1,952 family members of all ages in Ohio, found that respiratory illness incidence did not vary with stove type. More recently, Berwick and coworkers (190) followed 121 children for 3 months, 59 from homes with kerosene heaters and 62 from homes without such heaters. In a preliminary analysis of their data, they found that hours of heater use, which correlated strongly ($r = 0.70$) with 1-wk integrated NO_2 measurements, was significantly associated with the occurrence of illness lasting for 1 or more days.

The data concerned with lung function level in children are similarly inconclusive (table 7). Of the 4 investigations with large sample sizes (92, 186, 192, 193), 2 have demonstrated small but statistically significant effects of exposure to a gas stove (186, 192). In initial cross-sectional analysis of data from the Harvard Air Pollution Health Study, Speizer

TABLE 8

EFFECTS OF GAS COOKING ON PULMONARY ILLNESS, SYMPTOMS, AND FUNCTION OF ADULTS

Study Population	Outcome Measure	Results
441 upper-middle class families, including 1,054 adults over 15 yr, Ohio (184).	Incidence of acute respiratory illness, determined by biweekly telephone calls.	Respiratory illness incidence similar in homes using gas and electric stoves.
120 families from first study, including 269 adults over 18 yr, Ohio (185).	Incidence of acute respiratory illness, determined by biweekly telephone calls and validated by home visit.	Respiratory illness incidence similar in homes with gas and electric stoves.
1,724 adults, ages ≥ 20 yr, Maryland (195).	Major chronic respiratory symptoms, FEV_1, FVC.	Association between gas stove use and increased prevalence of respiratory symptoms, FEV_1 < 80% predicted, FEV_1/FVC < 70%, found in nonsmoking males only.
708 adults, ages ≥ 20 yr. Nonsmoking sample of above population (196).	Major chronic respiratory symptoms, FEV_1, FVC.	Significant association between gas stove use and increased prevalence of chronic cough and phlegm, low FEV_1/FVC.
102 nonsmoking women in lowest quartile of FEV_1 compared to 103 nonsmoking women in highest quartile, Michigan (121).	Comparison of proportions of cases and controls currently using gas stoves.	Marginal association between use of gas stove and lower lung function, (odds ratio = 1.8, p = 0.08).
97 nonsmoking adult females, Netherlands (194).	IVC, FEV, FVC, PEF, MEF_{75}, MEF_{25}, MMEF.	Cross-sectional analysis showed an association between current NO_2 exposure and decreases in most pulmonary function measures. No association with longitudinal decline in pulmonary function.

and associates (186) demonstrated average reductions, adjusted for parental smoking and socioeconomic status, of 16 ml and 18 ml in the FEV_1 and the FVC, respectively, in children from homes with gas stoves compared to children from homes with electric stoves. On expansion of the cohort, however, the reductions in FEV_1 and FVC, although still statistically significant, were 0.6% of predicted for the former and 0.7% for the latter (92). With adjustment for parental education, the effects of exposure to a gas stove were reduced by 30% and were no longer statistically significant. Cross-sectional analysis of lung function data collected at the children's second examination did not show significant effects of stove type. With extension of the follow-up interval, the investigators assessed determinants of pulmonary function growth and found no effect of gas stove exposure (114).

Hasselbad and associates (192) analyzed data from the Environmental Protection Agency's Community Health Environmental Surveillance System. They reported that in girls 9 to 13 yr of age, gas stove exposure decreased $FEV_{0.75}$ by an average of 18 ml after adjustment for parental education level and smoking status. An effect was not observed in girls 6 to 8 yr of age nor in boys 6 to 13 yr of age.

In another large cross-sectional study, Vedal and colleagues (193) examined the effects of stove type on spirometric volumes and flow rates in a sample of 3,175 children ages five to 14 years. With adjustment for parental smoking and socioeconomic status, exposure to a gas stove was not significantly associated with reduced lung function level.

The effects of gas stove exposure on lung function level were assessed in 5 other investigations, but the sample sizes were inadequate for detecting effects of the magnitude found in the larger studies. Keller and colleagues (184) performed spirometry on 1 occasion in a sample of the subjects in their surveillance study. The data were not reported separately for children, and overall there was no effect of stove type. In 1 of the cross-sectional surveys conducted in England, the investigators correlated lung function level with 1-wk measurements of NO_2 in the kitchen and in the childrens' bedrooms (180). With a sample of about 400 children, significant effects of NO_2 were not found. Dodge (188) and Ekwo and associates (91) did not find effects of stove type on lung function measures in their cross-sectional studies. Hosein and Corey (110) examined the influence of 9 indoor factors on $FEV_{1.0}$ in 1,357 nonsmoking white children from 3 U.S. towns. They preliminarily reported that exposure to gas stoves was significantly associated with a 0.148-L reduction in FEV_1 level in boys and 0.75-L in girls.

Only a few investigations provide data on acute and chronic effects of NO_2 exposure indoors on adults (table 8). Prospective studies of acute respiratory illness occurrence have not demonstrated excesses in residents of homes with gas stoves (184, 185, 197). Cigarette smoking and chronic respiratory diseases, potential confounding variables, were not considered in these studies.

Potential chronic effects have also been examined in populations of adults (table 8). Comstock and coworkers (195) reported that gas stove use was associated with a significantly increased prevalence of certain chronic respiratory symptoms and of ventilatory impairment in nonsmoking men, but not in smoking men or in women of either smoking status. A subsequent reanalysis limited to the never and former smokers showed significant increases in chronic cough and phlegm and in the prevalence of low FEV_1/FVC in association with gas stove use in both sexes (196).

In a study of 97 nonsmoking rural women from the Netherlands, personal exposure estimates were created by combining 1-wk measurements of NO_2 with time-activity information (118). The investigators demonstrated a cross-sectional association between lung function level and current NO_2 exposure but failed to show an association between retrospectively estimated exposure to NO_2 and longitudinal decline in pulmonary function during the antecedent 17 yr (194).

Using a case-control design, Jones and associates (121) compared cooking fuel exposures of 20- to 39-yr-old nonsmoking women in the highest and lowest quartiles of the lung function distribution in the Tecumseh Community Health Study. The odds ratio for the effect of cooking with gas on lung function level was 1.82 (p = 0.076).

Lebowitz and colleagues (124, 198, 199) have evaluated acute effects of gas stove exposure on lung function and symptoms

in 229 subjects drawn from 117 Tucson households. The families were sampled from a larger study population to include persons with and without asthma, allergies, and airway obstruction. During a 2-yr period, subjects completed symptom diaries and monitored their peak flow daily. Multivariate analyses indicated adverse effects of gas stoves on symptoms and peak flow rate in asthmatics but not in normal subjects (199). However, the magnitude of the effect is difficult to determine from the available publications.

Recently, Kasuga (200) proposed that the urinary hydroxyproline to creatinine ratio is a valid and sensitive indicator of lung damage from environmental pollutants, including tobacco smoke and NO_2. Hydroxyproline, an amino acid constituent of collagen, is a product of collagen catabolism; therefore, an increase in its excretion reflects an increase in collagen destruction.

Matsuki and associates (115, 201) conducted a cross-sectional study of 820 schoolchildren and their 546 mothers during both a summer and a winter period. They measured subjects' 24-h personal NO_2 exposures with filter badges and collected early morning urine samples for evaluation of the hydroxyproline to creatinine ratio. In multiple regression equations, passive smoking status and personal NO_2 were independent and significant predictors of this ratio in both schoolchildren and adult women in both seasons. Distance from a main road, as a surrogate for exposure to automobile exhaust, was found to be a stronger predictor of the ratio in summer than in winter in schoolchildren and a predictor only during the summer in adult women. A linear relationship was also found between the value of the ratio and the amount of passive exposure to tobacco smoke. Other studies, however, have not shown relationships of the hydroxyproline to creatinine ratio with either passive exposure to tobacco smoke (116) or with active smoking (117). Although the hydroxyproline to creatinine ratio could serve as a useful biochemical indicator of lung injury by NO_2 exposure, further investigations are needed to clarify ambiguities in the available data.

Definitive statements concerning the risk of NO_2 exposure from cooking with gas stoves cannot be made at present. Although many studies have examined respiratory illnesses, respiratory symptoms, and lung function in children and adults, their results are not consistent and are not adequate for establishing a causal relationship. Retrospective illness histories may be inaccurate and their results biased by whether the subjects have symptoms or illness at the time of interview (93). Variations in the characteristics of the study populations and differing endpoints may partly explain the differences among the studies. Confidence limits have not been uniformly presented in the studies on gas stoves, and the results of many of the smaller studies that have been judged as negative are probably consistent with the larger studies that show small effects.

Unfortunately, NO_2 exposures were directly measured in only a few investigations (180, 181, 191, 194), and in all of these the measurements spanned at most 2-wk periods. In the other studies, categorical variables, indicating gas or electric stove use, were employed. However, neither limited area measurements nor variables for stove type tightly predict actual personal exposure (170). Thus, the results of all investigations of the health effects of NO_2 exposure from gas stoves are affected by random misclassification. This type of bias reduces the magnitude of the observed association from the value that would be found if the exposure of subjects was correctly estimated (25). Ozkaynak and associates (202) have shown that misclassification introduced by the use of a categorical variable for stove type may introduce substantial underestimation of the true relative risk values associated with the actual NO_2 exposure.

Bias from inadequate control of confounding factors must also be considered in interpreting the foregoing studies (203). Confounding occurs when the effect of 1 variable on the outcome of interest has not been separated from the effects of other variables. For example, maternal smoking has been associated with reduced lung function level in children. Confounding by maternal smoking could arise in a particular study if mothers of infants living in homes with gas stoves were more likely to smoke. With regard to NO_2 exposure from gas stoves and effects on respiratory illnesses and symptoms, and pulmonary function in children, the potential confounding variables include parental smoking, socioeconomic status, and asthma. Active smoking, occupational exposures, and the presence of chronic respiratory diseases should also be considered in adults. Control of these potentially confounding factors has been variable among published studies (203), and in some studies socioeconomic status has been treated as a confounding factor. However, the effect of socioeconomic status represents a summation of the effects of associated environmental and familial factors, one of which may be gas stove exposure. Thus, control for socioeconomic status may reduce the likelihood of finding an effect of gas stove exposure.

Summary. The findings on NO_2 exposure and respiratory illnesses indicate that the magnitude of the NO_2 effect at concentrations encountered in most U.S. homes is likely to be small. Groups with particularly high exposures, such as the urban poor who heat with ovens and those who heat their homes with kerosene or gas space heaters, have not yet been adequately investigated. The evidence on respiratory symptoms and lung function level in children and adults is also inconclusive. However, because more than half of U.S. homes have gas cooking stoves and childhood respiratory illness is extremely common, even a small effect of gas stoves would assume public health importance. In order to detect associations of the anticipated small magnitude, future investigations should employ direct measurement of exposure, rather than surrogate variables. Infants and other potentially susceptible groups seem the most suitable populations for study. Nevertheless, the epidemiologic evidence implies that clinically relevant effects of NO_2 from gas stoves are uncommon at the concentrations found in most U.S. homes.

Carbon Monoxide

Introduction. Carbon monoxide is an odorless, colorless gas with well-characterized effects on oxygen transport (204). Carbon monoxide interferes with oxygen transport by avidly binding to hemoglobin to form carboxyhemoglobin and by shifting the oxyhemoglobin dissociation curve to the left. It also binds to myoglobin, but the physiologic significance of the formation of CO-myoglobin has not been established (205). Carboxyhemoglobin reduces oxygen delivery to tissues, as does the hypoxia of altitude. Tissues with the highest oxygen needs, myocardium, brain, and exercising muscle, are most affected by the formation of carboxyhemoglobin. Research on the health effects of lower levels of carbon monoxide exposure has emphasized consequences for these organs, particularly in subjects with diseases that make these organs vulnerable to reduced oxygen transport.

Exposure. Carbon monoxide has numerous sources in the home, the office, and other environments. In the home, emissions from gas appliances and cigarette

smoke, and from vehicles in attached garages may elevate CO levels. During cooking with a gas range, hourly CO concentrations typically range from 2 to 6 ppm and 1-h averages may exceed 12 ppm in conventional homes (28). One-hour CO concentrations in small apartments may reach twice the values in single-family residences. Use of gas stove for heating, a common practice among urban poor in northern climates, may increase CO concentrations to 25 to 50 ppm (206). Cigarette smoking is generally a minor source of CO in homes (64). Other combustion sources in homes are kerosene and gas space heaters (207–209).

Carbon monoxide exposure may also be received in vehicles, particularly when entry routes are available for CO from exhaust (210). During urban commuting, CO levels in cars may average 2 to 5 times the concentrations generally measured in homes and offices and by ambient air monitors (211–213). Offices may be contaminated by vehicle exhaust because of building design problems; high CO levels may result (214).

Health Effects. Most evidence on the health effects of low levels of exposure to carbon monoxide, as generally encountered in indoor environments, has been derived from experimental human exposures. This line of investigation has emphasized disease states that increase susceptibility to reductions of oxygen transport: coronary artery disease, peripheral vascular disease, and chronic obstructive pulmonary disease (204, 215, 216). While the evidence was once considered to indicate adverse effects of CO at low levels in affected persons, much of the data is now controversial.

Although the health effects of low levels of CO exposure are controversial, the problem of CO poisoning by indoor combustion sources has been well described and its dimensions should be recognized by clinicians. The clinical manifestations of CO poisoning primarily reflect the effects of reduced oxygen transport to organs, such as the heart and brain, with high oxygen demand. The neurologic manifestations range from impaired mentation and behavioral alterations to coma (217, 218). Delayed and persistent neurologic sequelae may follow CO poisoning (218). Cardiac effects include arrhythmias and myocardial infarction (217).

The nonspecificity and diversity of the manifestations of CO poisoning have been emphasized (217). In fact, the diagnosis of CO poisoning is frequently delayed while alternative diagnoses are considered. In a series from France, the most common misdiagnoses were food poisoning, psychiatric disorders, cerebrovascular disease, intoxication, and heart disease (219). The finding of retinal hemorrhages on fundoscopic examination should alert the clinician to possible CO poisoning (220, 221). Kelly and Sophocleus (220) reported 12 cases of subacute CO poisoning; retinal hemorrhages were found in each of the 5 patients exposed more than 12 h. The incidence of CO poisoning may rise with increased use of space heaters and woodstoves.

Summary. Carbon monoxide poisoning is a well-documented clinical entity that follows exposure to high levels of CO. Effects of the lower levels of CO exposure generally encountered in indoor environments are controversial at present.

Woodsmoke

Introduction. Since the 1973 oil embargo, there has been a resurgence of residential wood use in the United States. During the decade of the 1970s, the shipment of woodstoves increased 10-fold and the current inventory of woodstoves is estimated to exceed 11 million (222). Residential woodburning typically occurs under oxygen-starved conditions that increase emission rates for CO, respirable particulates, and polycyclic aromatic hydrocarbons. In many communities where woodburning is common, ambient concentrations of these pollutants have increased as a result (223). The use of fireplaces and stoves may potentially result in increased indoor concentrations of smoke components by reentrainment of outdoor air or by direct leakage into indoor air.

Exposure. Few assessments of the impact of woodburning stoves and fireplaces on indoor air quality have been performed. Limited evidence suggests that the rate of pollutant emissions from a wood-burning source depends primarily on the degree of air-tightness of the source. Under proper operating conditions the newer "airtight" residential woodstove is under negative pressure and should not leak combustion by-products into the home. However, under non-airtight operations and during startup, stoking, and reloading, pollutants can be emitted indoors. Traynor and colleagues (224) reported indoor CO concentrations of 0.4 to 2.8 ppm during operation of "airtight" stoves, whereas average levels of 1.8 to 14 ppm occurred during operation of "non-airtight" stoves. For submicron sized particles, indoor concentrations were slightly above background (zero to 30 μg/m^3) during the use of "airtight" stoves and substantially higher with the "non-airtight" stoves (200 to 1,900 μg/m^3). Indoor concentrations of 5 polycyclic aromatic hydrocarbons greatly exceeded outdoor levels when the "non-airtight" stove was used (224).

These results are consistent with the findings of a study of personal exposures to respirable particulates in a rural community with substantial woodburning for winter heating (225). Analysis of respirable particulate data collected over 7 days of sampling in 24 homes in Waterbury, Vermont, suggested that homes with airtight woodburning stoves have about 4 μg/m^3 higher indoor concentrations than do the homes without woodburning stoves (225). The elemental composition of indoor and outdoor particles was examined for 5 of these homes. Using the elements as tracers for wood, automobile exhaust, and other sources of particles, as well as for measuring penetration of ambient air, the investigators confirmed that the increased indoor particle levels were due to woodburning.

Elevated concentrations of pollutants may also be caused by woodburning in fireplaces. Moschandreas and colleagues (63, 226) reported benzo(a)pyrene and respirable particulate levels indoors and outdoors from a series of measurements made in 3 homes, 2 with fireplaces and the third with a woodstove. The outdoor concentrations of benzo(a)pyrene rarely exceeded 1 ng/m^3. The indoor benzo(a)pyrene concentrations were substantially higher than outdoors on days when the woodstove was used, averaging 4.7 ng/m^3 indoors. Benzo(a)pyrene was only measured on 1 woodburning day for 1 home with a fireplace. On this day, the integrated particle samples indoors exceeded 11 ng/m^3 benzo(a)pyrene, while those outdoors were less than 0.5 ng/m^3. Respirable particulates were also elevated in all 3 residences on woodburning days. Levels ranged from 14.3 to 72.5 μg/m^3 in the home with the woodstove, and were 159.9 and 67.6 μg/m^3 on 1 woodburning day in each of the homes with a fireplace. The investigators concluded that woodburning in a stove or a fireplace may be an important source of indoor pollution.

In summary, airtight woodstoves contribute relatively low concentrations of particulates, CO, and polyaromatic hydrocarbons to the indoor environment. Woodburning in fireplaces and non-

airtight woodstoves may lead to substantially higher levels of these pollutants. Nonetheless, woodsmoke is a complex mixture and only a few of its components have been measured in homes. For example, measurements of aldehydes such as acrolein have not been performed during residential woodburning. However, the characteristic smell of woodburning in stoves and fireplaces indicates that odorous compounds, some of which are most likely aldehydes, are released by combustion of wood. Further assessment of the impact of woodburning on indoor air quality is needed to describe fully the range of contaminants produced and their concentrations under differing conditions of operation and combustion.

Health Effects. Limited data have been published concerning the health effects of residential wood combustion. *In vitro* experiments demonstrate that emissions from a woodstove induce sister chromatid exchange (227) and are mutagenic, as assessed by the Ames Salmonella assay (228). Using a rabbit model, Fick and colleagues (229) studied the effects of woodsmoke on pulmonary macrophages. They reported that smoke-exposed rabbits, in comparison with controls, produced significantly more cells of all types on bronchoalveolar lavage, and that the rabbit macrophages exhibited a decrease in adherence, phagocytic rate and bacterial uptake. Macrophage viability and bactericidal processing were not affected. Wong and coworkers (230) evaluated the response of guinea pigs to woodsmoke with repeated CO_2 challenges. After exposure, respiratory frequency decreased and ventilatory response to CO_2 was diminished. These effects were transient and full recovery occurred within 3 days.

Only a few epidemiologic studies on the health effects of woodsmoke have been performed. Studies from less developed countries indicate an association between intense smoke exposure in dwellings and chronic pulmonary disease. In a house-to-house survey of adults more than 20 yr of age in Nepal, Pandey (231) found that chronic bronchitis was equally prevalent in men and women, affecting 18.9%, in contrast to the male preponderance usually observed. Further analysis of the data demonstrated an association between prevalence rates for chronic bronchitis and domestic smoke exposure as measured by the number of hours spent daily near the stove (232). Pandey and colleagues (233) subsequently evaluated respiratory function of 150 women ages 30 to 44 yr from 2 rural villages in Nepal. In cigarette smokers, spirometric test results worsened as reported hours of smoke exposure increased, but a similar effect was not present in nonsmokers.

Master (234) randomly selected 94 New Guinea residents for a health evaluation that included a complete history and physical examination. The prevalence of clinical symptoms or abnormal pulmonary findings was extremely high at all ages; 90% of subjects 40 yr of age and older were affected. Although Master collected only descriptive clinical data and no information on exposures, he attributed the high prevalence of abnormalities to domestic smoke exposure. Based on the findings of a cross-sectional study, Anderson (235) has also suggested that woodsmoke exposure contributes to the development of chronic lung disease in adults in New Guinea.

Respiratory effects of woodsmoke have also been examined in children from less developed countries. Anderson (236) conducted a cross-sectional study and a longitudinal study to assess the effects of woodsmoke pollution on children in New Guinea. He evaluated 1,650 children drawn from 2 contrasting communities, 1 at sea level where wood was not burned and 1 in the highlands where wood was commonly burned. The 2 groups did not differ on spirometric testing, physical examination, or clinical history. He also followed 112 children with differing levels of woodsmoke exposure and did not find a consistent relationship between exposure and respiratory abnormalities during a 30-wk surveillance period. In contrast, Kossove (237) reported that Zulu infants less than 13 months of age with severe lower respiratory tract diseases were twice as likely to have a history of daily heavy smoke exposure as were infants without such disease.

Although these studies implicate domestic smoke exposure as a risk factor for the development of respiratory disease in less developed nations, their results should not be generalized to more developed nations. The exposures are orders of magnitude lower on average in more developed countries than in less developed countries. In the less developed countries, low efficiency stoves are used for long periods of time in small huts with poor ventilation, and agricultural waste and dung are often used as fuel (238). These conditions may lead to particulate and benzo(a)pyrene levels that are 10 to 100 times higher than those found in U.S. homes with woodburning stoves (239).

Data on health effects of residential wood combustion in the United States are sparse. In a case report, Honicky and colleagues (240) described an infant with recurrent hospitalizations for severe lower respiratory tract disease characterized by wheeze and pneumonia. The child improved when hospitalized and then relapsed within 12 h after returning home. After the parents removed their woodstove, the child's illnesses ceased. This case prompted the investigators to conduct a prevalence study of respiratory symptoms in 62 children in Michigan, 31 from homes with and 31 from homes without woodburning stoves (241). Using a standardized questionnaire, interviewers asked parents about their children's respiratory symptoms during the previous winter. Symptoms were classified as present or absent and as mild, moderate, or severe. The proportion of children with moderate or severe symptoms was much greater in the group from homes with woodstoves: 84% of children in this group reported at least 1 severe symptom as compared to 3% of the control group. Parental smoking and socioeconomic status were similar in both groups.

In a study of similar design in Massachusetts, Tuthill (242) retrospectively ascertained episodes of acute respiratory illnesses from January through March from 399 parents of school-age children. In contrast to Honicky's results, use of a woodburning stove was not associated with chronic respiratory disease, symptoms such as fever, sore throat, rhinitis, cough and wheeze, or excess (more than 1) respiratory illness. Differences in study populations, type of wood burned or ascertainment of illness may explain the conflicting results of these studies.

Another potential hazard of woodburning stoves is illustrated by a recent case report of a Wisconsin family that experienced arsenic poisoning (243). Over a 3-yr period, the family displayed a variety of symptoms ranging from rashes and muscle cramps to seizures and loss of consciousness. An environmental evaluation of their house revealed that they were burning plywood treated with a chromium-copper-arsenate mixture in their stove.

Summary. Woodsmoke is a complex mixture of gases and particles that has a wide range of potential respiratory effects. The unconfirmed observations of Honicky and colleagues (241) that woodsmoke causes acute respiratory illnesses and symptoms in U.S. children require further study. Investigations in less developed countries suggest that domestic

smoke exposure contributes to the development of chronic lung disease. This important hypothesis cannot be tested with sufficient sensitivity in most populations in the United States but should be pursued in appropriate locales. Recurrent severe respiratory disease with no underlying causes in an infant should prompt the clinician to determine whether a woodstove is present in the home. In these situations, a therapeutic trial of discontinuing its use seems warranted.

Addendum

During 1987, several new sources of information on indoor air pollution and health have been published. The 4th International Conference on Indoor Air Quality and Climate was held in August 1987. The proceedings were published by the Institute for Water, Soil and Air Hygiene in Berlin (mailing address: Institut für Wasser-, Boden- und Lufthygiene des Bundesgesundheitsamtes, Corrensplatz 1, D-1000 Berlin 33). The U.S. Environmental Protection Agency report "EPA Indoor Air Quality Implementation Plan" and its appendices provide a comprehensive review. Two new reports on environmental radon are available: "Lung Cancer Risk from Indoor Exposure to Radon Daughters," Publication 50 of The International Commission on Radiological Protection, and the report of the Biological Effects of Ionizing Radiation (BEIR) IV Alpha Committee of the National Academy of Sciences.

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State of Art

Health Effects and Sources of Indoor Air Pollution. Part II[1-3]

JONATHAN M. SAMET,[4] MARIAN C. MARBURY,[5] and JOHN D. SPENGLER

Contents

Health Effects of Indoor Air Pollution (Cont'd)

Biological Agents

Introduction. Numerous and diverse biological agents that cause human disease are present in indoor air. Mechanisms of disease pathogenesis are now well understood for many of these agents; they produce illness primarily through infection of the respiratory tract and through immune responses. Because of the diversity of these agents and their associated illnesses, this review will address only selected and illustrative examples of the health effects of biological agents in indoor air. More detailed treatments are included in the National Research Council's report on indoor air (15) and in other recent publications (19–24). With regard to immunologically mediated diseases, we review hypersensitivity pneumonitis and humidifier fever and the role of selected biological agents in the etiology and exacerbation of asthma. We will also briefly consider 2 problems of infection associated with indoor air: the transmission of Legionnaires' disease and infection with *Aspergillus* through contamination of hospital and office environments.

Exposure to Biological Agents. Myriad biological agents may contaminate the air within a home, office, or other indoor environment. The most prevalent are viruses, bacteria, actinomycetes, fungal spores, algae, amoebae, arthropod fragments and droppings, and animal and human dander (244, 245). In homes and in other environments, moisture is critical for the growth of microorganisms. Humidifiers, air conditioning systems, and areas of water damage may provide a suitable environment for proliferation of microorganisms. The initial colonization may be from outdoor or indoor sources or from organisms in the water.

Most bacteria in indoor air originate from humans, whereas most fungi in indoor air originate from spores from outdoor sources (15, 245). Many indoor environments provide sufficient moisture and an appropriate temperature for the growth of fungi, bacteria, mites, and other biological agents. Moist surfaces of leather, wood, and plaster, soaps, greases and some oils, cloth fabrics, paper, and some pastes and glues can support growth of microorganisms, as can wicker baskets and chairs, and ornamental plants. Moist locations within a home include bathrooms, damp or periodically flooded basements, and areas with water leaks. The evaporation pans of refrigerators, shower heads, and hot tubs have also been identified as possible sources of bacteria (245).

Concentrations of microorganisms in the indoor environment have not been well-characterized. Standardized sampling methods have not been developed,

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and collection efficiency for biological particles would be expected to vary with particle size and density and with the aerodynamics of the sampling method. Further variation in concentration will arise from cycles of proliferation and of the physical forces that place organisms into the air. Thus, reports on concentrations as numbers of colony-forming units (cfu) per cubic meter cannot be interpreted without knowledge of the specific species, the efficiency of collection by the sampling apparatus, and the conditions under which sampling was performed.

Hypersensitivity Pneumonitis and Humidifier Fever. In 1970, Banaszak and coworkers (246) reported that 4 of 27 workers in one office had developed hypersensitivity pneumonitis from exposure to thermophilic actinomycetes contaminating an air-conditioning system. Subsequently, hypersensitivity pneumonitis and humidifier fever, an influenza-like syndrome without prominent pulmonary manifestations, have been described in association with contaminated air treatment systems in offices, homes, and automobiles (247–249).

A wide range of biological sources for potentially sensitizing antigens has been described, including thermophilic actinomycetes, diverse fungi, bacteria, amoebae, and nematodes. In some outbreaks, a specific antigenic exposure underlying the illness could not be identified (250–253). The offending antigens have been introduced into indoor environments through central and room humidifiers (252, 254–257), contaminated heating and cooling systems (246, 251, 258, 259), moisture-damaged building materials (260), cool mist vaporizers used in the home (250), and automobile air conditioning systems (261).

The literature on hypersensitivity pneumonitis and humidifier fever consists primarily of individual case reports and small series from identified outbreaks. While these studies do not address the overall prevalence and incidence of these illnesses, they do provide comprehensive clinical descriptions. As with hypersensitivity pneumonitis, associated with other exposures, both acute and chronic forms may result from exposure to indoor antigens (252). Persons with the acute form characteristically have fever, chills, cough, and dyspnea after exposure. In the more chronic form, patients may present with progressive dyspnea and lung function impairment. The diagnosis of either form is based on the clinical history, evidence of exposure, the presence of precipitating antibodies to environmental antigens, response to inhalation challenge, and improvement with cessation of exposure. Precipitating antibodies may be present in exposed but unaffected persons, however.

Asthma. Both outdoor and indoor air pollutants have long been considered as important in the etiology and exacerbation of asthma. In a home, components of house dust, animal proteins, and fungal spores may provoke asthma through immediate hypersensitivity (262–264). In an office, aeroallergens and irritant agents may be present. Pollens and molds from outdoor sources may penetrate into the home, office, and other environments.

While qualitative and quantitative aspects of aeroallergens in indoor air have been well described, their contribution to asthma has been more difficult to characterize. Epidemiologic studies have been conducted to examine the relationship between the severity of asthma and exposure to aeroallergens and other indoor pollutants (for example, see 198, 199). However, the results of these studies are limited by the difficulties of monitoring personal exposures to pollutants and of separating the effects of the many factors that influence asthma's severity. A detailed presentation of these studies is beyond the scope of the present review. Comprehensive discussions of the aeroallergens in indoor air can be found in the National Research Council's report on indoor pollutants (15) and in recent reviews by Reed and coworkers (263) and Ausdenmoore and Fischer (264).

We briefly consider the data on house dust mites and asthma because this antigen has been intensively investigated and the literature is illustrative. House dust mites live in mattresses and furniture stuffing, and their numbers tend to increase with the environment's humidity (264, 265). The mite, *Dermatophagoides pteronyssinus*, has been shown to be highly prevalent in homes in Europe, and this species as well as *Dermatophagoides farinae* have been found in houses in the United States (266–268). The major allergen of *D. pteronyssinus* has been designated as antigen P_1. This potent allergen may be found in high concentrations in dust from beds and floors; airborne levels in homes with undisturbed dust are quite low but increase with domestic activity (269, 270).

Clinical studies provide convincing evidence that inhalation of house dust contaminated with mites causes asthma (271). However, the prevalence of house-dust-mite-related asthma has not been established through appropriate population-based studies. Increased exposure has been associated with a greater risk of asthma in adults (272). Preventive measures, including frequent cleaning, removal of carpeting, pillows, and quilts with feathers, and covering mattresses with plastic reduce the concentrations of mites in house dust (271, 273). Some clinical trials suggest benefits from these measures in children and adults, although the studies are not uniformly positive (274–279).

Legionnaires' Disease. Legionnaires' disease refers to acute bacterial infection with *Legionella pneumophila.* The clinical features and many aspects of the epidemiology of Legionnaires' disease have been well characterized (280). Both epidemic and sporadic cases may result from contamination of indoor air with *Legionella pneumophila.* Because Legionnaires' disease exemplifies the spread of an infectious illness by air treatment systems, we will consider representative outbreaks.

Legionella pneumophila has been isolated from water sampled from cooling towers and evaporative condensers, devices used to cool water for buildings (281). Failure to treat the water with appropriate disinfectants may permit the growth of microbial agents, including the Legionnaires' disease bacterium. Outbreaks of Legionnaires' disease have been described in association with contaminated air treatment equipment in hospitals and offices. The 1976 epidemic in Philadelphia has been attributed to airborne transmission of the bacterium, although its source has never been identified (282). More convincing evidence of airborne transmission has been obtained from other outbreaks, however. Dondero and coworkers (283) described a 2-month-long outbreak in those in contact with a single hospital as patients, visitors, employees, or passersby. *Legionella pneumophila* was present in water in an auxiliary cooling tower in use during this time; the aerosol that drifted away from the tower entered the hospital's ventilation system and also dropped into the street below.

In an outbreak in a new sealed office building in San Francisco, at least 14 of 1,000 workers developed Legionnaires' disease over a 2-wk period (284). *Legionella pneumophila* was grown from water samples taken from the building's air-conditioning cooling tower, and further cases were not observed after the

cooling tower was disinfected. Airborne spread of the bacterium within single buildings has probably led to other outbreaks as well (285–288).

Nosocomial cases of Legionnaires' disease have also been attributed to aerosols generated by respiratory therapy devices (289). Hospital water supplies may become contaminated with *Legionella pneumophila* (290, 291), and the organism has been cultured from plumbing fixtures, such as shower heads (292–294). The mechanism by which pneumonia is acquired from these waterborne organisms has not been identified, but aerosolization and contamination of indoor air seem plausible.

Aspergillus *Infections*. Nosocomial infections with *Aspergillus* species also illustrate the potential for disease transmission through ventilation systems. Patients with defects of cell-mediated immunity are particularly vulnerable to infection by these organisms. Outbreaks of *Aspergillus* infection have been described in hospitalized patients in association with airborne spread related to inadequate ventilation systems (295), contaminated ventilation systems (296, 297), improperly functioning ventilation systems (298), construction activity (299), and building materials (300).

Formaldehyde

Introduction. The recent recognition of numerous sources of formaldehyde in indoor environments has raised widespread concern about the health hazards of this pollutant. A colorless volatile gas with a characteristic odor, formaldehyde is highly soluble in water and thus irritating to the mucous membranes of the eyes and upper respiratory tract. The public first became aware of possible health effects of formaldehyde through reports that residents of homes insulated with urea formaldehyde foam insulation (UFFI) experienced a wide variety of symptoms that were attributed to release of formaldehyde from this material. Although use of UFFI has virtually ceased in the United States and Canada, concern remains about the health effects of formaldehyde because of its widespread use in industrial processes, building materials, and consumer products. The 1981 report of the Committee on Aldehydes of the National Research Council (301) and the 1983 report of the Consensus Workshop on Formaldehyde (302) provide recent assessments of formaldehyde's toxicity, and L'Abbe and Hoey (303) have reviewed the health effects of UFFI.

Exposure. Formaldehyde has many sources in the home: paper products, floor coverings, carpet backings, adhesive binders, permanent-press clothing, tobacco smoking, combustion processes, resins, and cosmetics. Particularly high concentrations may result from the use of UFFI, a resin of urea, formaldehyde, and water. UFFI can be used to insulate already constructed homes and as many as 200,000 homes in the United States may have been insulated with this material in 1980 (15). After installation UFFI releases formaldehyde for a short time as it hardens. If the UFFI is improperly cured, release of formaldehyde may be sustained and may take place in large quantities.

Godish (304) monitored for formaldehyde in residences with and without UFFI. Concentrations in the homes with UFFI ranged from 0.02 to 0.13 ppm, whereas in the homes without UFFI they ranged from 0.03 to 0.07 ppm. Georghiou and coworkers (305) measured formaldehyde in 44 homes with UFFI and in 6 control homes. The homes with UFFI more recently installed tended to have higher concentrations. Eighty-four percent of the repeated measurements in such homes exceeded 0.06 ppm. In homes with UFFI installed more than 3 yr prior to monitoring, only 22% of the formaldehyde concentrations exceeded 0.06 ppm, whereas in homes with UFFI installed more recently, 58% of the samples exceeded 0.1 ppm.

Mobile homes are constructed with large quantities of particle board, which is manufactured with formaldehyde-containing adhesive binders. As a result of this construction method, formaldehyde concentrations tend to be higher in mobile homes than in conventional homes. In several studies, formaldehyde levels have been surveyed in mobile homes and have been found to greatly exceed those found in conventional homes. In a survey of mobile homes in Texas, Stock and coworkers (306) reported integrated concentrations ranging from less than 0.02 to 0.78 ppm. Dally and colleagues (307) studied mobile homes whose residents were concerned about formaldehyde exposure and found that 50% of the values were greater than 0.47 ppm and that the range was as high as 3.6 ppm.

Numerous potential sources of formaldehyde exist in office buildings, including insulation, new furniture and furnishings, carpets, carbonless copy paper, and cigarette smoke. Formaldehyde has been measured infrequently in office buildings, and then usually as part of an evaluation of building-related illness. Breysse (308), summarizing data from 20 health hazard evaluations conducted in Washington, reported that concentrations ranged from 0.01 to 0.30 ppm. While formaldehyde has been implicated as the causative agent in about 4% of episodes of building-related illness investigated by the National Institute of Occupational Safety and Health (309), concentrations in these episodes have been well below the permissible exposure level established by the Occupational Safety and Health Administration.

Health Effects. Both acute and chronic health effects have been associated with formaldehyde exposure. A variety of short-term signs and symptoms are commonly accepted as causally related to exposure; some occur at levels that have been measured in residential air (table 9). The wide range of concentrations at which individual symptoms occur suggests a large variation in individual sensitivity to formaldehyde. As would be an-

TABLE 9

ACUTE HUMAN HEALTH EFFECTS OF FORMALDEHYDE AT VARIOUS CONCENTRATIONS*

Reported Effects	Formaldehyde Concentration (ppm)
None reported	0.0–0.5
Neurophysiologic effects†	0.05–1.5
Odor threshold	0.05–1.0
Eye irritation‡	0.01–2.0
Upper airway irritation	0.10–25
Lower airway and pulmonary effects	5–30
Pulmonary edema, inflammation, pneumonia	50–100
Death	> 100

* Adapted from table 7-2 in reference 301.
† As measured by determination of optical chronaxy, electroencephalography, and sensitivity of dark-adapted eyes to light.
‡ The low concentration (0.01 ppm) was observed in the presence of other pollutants that may have been acting synergistically

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TABLE 10

SURVEYS OF OCCUPANTS LIVING OR WORKING IN MOBILE HOMES OR HOMES WITH UFFI

Study Population	Findings (%)	Comments
424 adults, 99 children living in 334 mobile homes. Complaint investigations,* Washington State (310)	Eye irritation: A, 58; C, 41 Throat irritation: A, 66; C, 62 Chronic headache: A, 40; C, 16 Chronic cough: A, 9; C, 33 Memory lapse/drowsiness: A, 24; C, 7	Formaldehyde levels: 0.03 to 1.77 ppm; no control group; exposure-response not examined
256 adults and children living in 65 mobile homes or 35 other structures. Complaint investigations,* Wisconsin (307)	Eye irriation: 68 Throat irritation: 57 Headache: 53 Cough: 51 Difficulty sleeping: 38 Wheezing: 20	Formaldehyde levels: 0.0 to 3.68 ppm; no control group; exposure-response not examined
162 residents of 68 homes with UFFI. Complaint investigations,* Connecticut (311)	Eye irritation: 39 Nose/throat/lung irritation: 48 Headache: 17 No apparent relationship between symptoms and crude formaldehyde level	Formaldehyde levels: 0.0 to 10 µg/L, with detectable and nondetectable levels
Unknown number of residents in 443 families living in mobile homes. Complaint investigations,* Texas (312)	No difference in symptom prevalence in families living in homes with and without detectable levels	Formaldehyde levels: 0.0 to 8 ppm; comparison of homes with detectable and nondetectable levels
1,396 residents of UFFI homes; 1,395 residents of non-UFFI homes. Retrospective cohort, New Jersey (313)	Exposed more likely to report wheezing than nonexposed: Wheezing: Exposed, 0.6 Nonexposed, 0.1 Burning skin: Exposed, 0.7 Nonexposed, 0.1 Subgroup in whose homes odor persisted > 7 days after foam installed, had higher symptom incidence	Population-based study: formaldehyde concentrations not measured
70 exposed employees of 7 mobile home care centers; 34 nonexposed employees of 3 permanent structures, Denmark (314)	Exposed reported significantly more symptoms than did nonexposed. Menstrual irregularities: Exposed, 35 Nonexposed, 0 Excessive thirst: Exposed, 60 Nonexposed, 5 Eye irritation: Exposed, 55 Nonexposed, 15 Headache: Exposed, 80 Nonexposed, 50	Formaldehyde levels in mobile day care centers: 0.24 to 0.55 ppm; permanent structures: 0.05 to 0.11 ppm
21 exposed workers in mobile home office, 18 nonexposed workers in another office, Illinois (315)	Exposed reported significantly more symptoms. Eye irritation: Exposed, 81 Nonexposed, 17 Throat irritation: Exposed, 57 Nonexposed, 22 Fatigue: Exposed, 81 Nonexposed, 22 Headache: Exposed, 76 Nonexposed, 11 No difference in pulmonary function	Formaldehyde levels in offices ranged from 0.12 to 1.6 ppm

Definition of abbreviations: A = adults; C = children.

* Complaint investigations were instigated at residents' requests.

ticipated from the high water solubility of formaldehyde, acute mucous membrane and eye irritation are the most commonly reported symptoms in residents of mobile homes and homes insulated with UFFI (table 10). Many questions still remain, however, concerning other acute and chronic health effects of formaldehyde: human carcinogenicity, nonmalignant effects on the respiratory tract, and neurobehavioral impairment. Data on these issues derive largely from surveys of residents of mobile homes and homes insulated with UFFI (table 10), and from epidemiologic and clinical investigations of occupationally exposed workers.

Cancer. In 1979, the Chemical Industry Institute of Toxicology reported that rats exposed to formaldehyde developed nasal cancer, a tumor rarely found in control animals (316). This malignancy developed in 103 of 206 rats exposed to a concentration of 14 ppm and 2 of 235 rats exposed at 5.6 ppm. This first report of formaldehyde carcinogenicity in an animal model, which has subsequently been independently replicated (317), stimulated the rapid performance of epidemiologic investigations (table 11).

Because of its high water solubility, formaldehyde is primarily deposited in the upper respiratory tract, and cancer in this region is of primary concern. Halperin and coworkers (329) reported a case of nasal cancer in a worker exposed to formaldehyde for 25 yr in the textile-finishing industry. However, epidemiologic studies of mortality among formaldehyde-exposed professional and industrial groups have not provided consistent evidence of an association between formaldehyde exposure and upper respiratory tract cancer.

Retrospective cohort studies of formaldehyde-exposed workers have not shown an excess of nasal cancer (table 3) (323, 325–328). However, even the largest of these cohort studies (327) had only limited statistical power (80%), as calculated by the investigator, to detect a fourfold increase in risk for this rare cancer. Additionally, follow-up periods in these studies may have been too short if nasal cancer does not occur in excess until long after first exposure.

Several case-control studies of the association between formaldehyde exposure and nasal cancer have now been performed. In a hospital-based case-control study in the United States, Brinton and coworkers (330) demonstrated an association between nasal cancer and previous employment in the textile industry, an industry in which use of formaldehyde is widespread. They also found, however, that cases reported a history of formaldehyde exposure less frequently than did control subjects. A population-based case-control study of nasal cancer in Norway failed to show an association with occupations classified as involving potential exposure to formaldehyde (331), although the study was not originally designed to investigate formaldehyde's role. Because study subjects had not been asked directly about previous formaldehyde exposure, an industrial hygienist evaluated the occupational histories collected by interview and made a judgment on the likelihood of exposure for each subject. Vaughan and colleagues (332) employed a similar method of exposure assessment in a population-based study in western Washington. No association was detected between jobs with potential formaldehyde exposure and cancers of the pharynx, sinus, or nasal cavity.

In contrast, 2 other case-control studies have demonstrated a positive association between nasal cancer and potential formaldehyde exposure. In a recent Danish case-control study of nasal cancer, a list of patients with nasal can-

TABLE 11

STUDIES OF FORMALDEHYDE-EXPOSED COHORTS AND CANCER

Study	Findings	Comments
Cohort study of pathologists, Great Britain (318)	SMR elevated for lymphoma and hematopoietic neoplasms (211) but not for leukemia	Less than 10% of cohort deceased, less than 20 yr of follow-up
Proportional mortality study of embalmers, New York (319)	PMR significantly elevated for cancers of skin (221) and colon (143); nonsignificantly for cancers of brain (156) and kidney (150), and leukemia (140)	
Proportional mortality study of embalmers, California (320)	PMR significantly elevated for cancers of colon (188), brain (191), and prostate (176), and leukemia (174); nonsignificantly for bladder cancer (138)	
Cohort study of pathologists, Great Britain (321)	SMR significantly elevated for brain cancer (300) but not for lymphoma	Less than 5% of cohort deceased; 6 yr of follow-up
Cohort study of anatomists, USA (322)	SMR elevated for brain cancer (271, 95% CI = 130–499) and leukemia (148, 95% CI = 71–272)	Excess brain cancer persisted when psychiatrists used as a reference group
Cohort study of undertakers, Canada (323)	SMR nonsignificantly elevated for brain cancer (115) and leukemia (160)	20 yr of follow-up
Proportional mortality study of chemical plant employees, Massachusetts (324)	PMR nonsignificantly elevated for cancers of digestive organs (152) among formaldehyde-exposed workers. No data reported on brain cancer and leukemia	No evidence of trend of mortality in relation to exposure
Cohort study of chemical plant employees, USA (325)	SMR significantly elevated for cancers of genitourinary tract (169) SMR for leukemia not elevated No data for brain cancer	Case-control study within cohort showed no association between GU cancer and a general plant exposure
Cohort study of chemical plant employees, Great Britain (326)	SMR for lung cancer significantly elevated (124) in 1 of 6 men most highly exposed	Retrospective assessment made of level of exposure
Cohort study of industrial workers with formaldehyde exposure, USA (327)	SMR significantly elevated for nasopharyngeal cancer (318). SMR nonsignificantly elevated for lung cancer (111) and Hodgkin's disease (142)	Largest study reported to date, retrospective assessment of exposure level
Cohort study of garment workers, USA (328)	SMR significantly elevated for buccal cavity (343) and connective tissue cancer (364)	Retrospective assessment of exposure level

Definition of abbreviations: SMR = standardized mortality ratio, PMR = proportional mortality ratio

cer ascertained through a cancer registry was linked to employment data from a national pension fund that has covered all employees in Denmark since 1964 (333). Exposure was determined using a method similar to that employed in the Norwegian study. The investigators reported an association between nasal cancer and jobs with potential for formaldehyde exposure (relative risk = 2.8, for both males and females; 95% confidence interval = 1.8 to 4.3 for males and 0.5 to 14.3 for females). Adjustment for exposure to wood dust, which is associated with both nasal cancer and formaldehyde exposure, decreased the relative risk to 1.6 for males (95% confidence intervals = 0.7 to 3.6).

Hayes and coworkers (334) in the Netherlands studied 91 men with cancer of the nasal cavity and paranasal sinuses and 195 male control subjects. Potential formaldehyde exposure was assessed independently by 2 industrial hygienists who reviewed job histories obtained by interview. The relative risk for the association between formaldehyde exposure and nasal cancer was 1.9 or 2.5, depending on which industrial hygienist's assessment was used. The relative risk was highest for men with squamous cell carcinoma who had any exposure to formaldehyde but little or no exposure to wood dust (relative risk = 1.9 or 3.0).

Although the majority of studies performed to date have considered only occupational sources of formaldehyde exposure, a study of the relationship between residential formaldehyde exposure and nasal cancer has recently been reported. Vaughan and coworkers (335), in addition to obtaining job histories, also inquired about subjects' residential exposures to formaldehyde, including whether the subjects had ever lived in a mobile home. No association was found between residence in a mobile home and cancers of the oropharynx or hypopharynx or of the sinus and nasal cavity. However, an increased risk of nasopharyngeal cancer was associated with living in a mobile home. Residence in a mobile home for 1 to 9 yr was associated with a relative risk of 2.1 (95% confidence interval = 0.7, 6.6); the risk increased to 5.5 (95% confidence interval = 1.6, 19.4) when residence exceeded 9 yr. Although based on only 8 exposed cases of nasopharyngeal cancer, the association persisted after control for confounding by cigarette smoking and race.

Although an excess of nasal cancer has not been demonstrated in cohort studies of formaldehyde-exposed industrial workers, 2 recent studies provide evidence of a possible relationship between formaldehyde and buccal-pharyngeal cancer. Blair and coworkers (327) studied more than 26,000 workers employed in 10 different plants where formaldehyde was either used or produced. They reported a statistically significant excess of nasopharyngeal cancers (SMR = 318) and a nonsignificant excess of oropharyngeal cancer (SMR = 192), although there was no overall excess of buccal-pharyngeal cancers. Excesses of buccal cavity (SMR = 343), but not pharyngeal (SMR = 113) cancer, were noted by Stayner and colleagues (328) in a cohort study of garment workers exposed to formaldehyde. Unlike rats, humans breathe through their mouths as well as their noses. Thus, the buccal cavity may be a biologically plausible site for formaldehyde-induced cancer in humans.

The occurrence of lung cancer has also been examined in the formaldehyde-exposed cohorts, and an excess of lung cancer has been reported for 2 of the populations. Acheson and coworkers (326) studied 7,000 men employed in 6 different chemical and plastics factories. They found a 24% increase in lung cancer in one of the factories when national mortality rates were used as the standard of comparison but no significant increase when local rates were used. However, the lung cancer risk was greatest among men who started employment between 1935 and 1946, when exposures were highest. Additionally, the standardized mortality ratio was elevated only among men in the high exposure category. Blair and colleagues (327) reported a small and nonstatistically significant excess of lung cancer (the SMR was 111 for exposed workers, 93 for nonexposed workers). There was a statistically significant 32% increase among workers with more than 20 yr since first exposure. However, the investigators discounted the significance of this finding, noting that the excess did not increase with estimates of intensity or duration of exposure, or with cumulative exposure. Other studies have not found excess lung cancer in formaldehyde-exposed populations (319–324).

Cancers of other sites have also been examined in these investigations. Studies of embalmers (319, 320, 323), anatomists (322), and pathologists (318), but not of formaldehyde-exposed industrial workers (325–328, 336), have demonstrated significant excesses of brain cancer; excessive leukemia has also been found in embalmers (319, 320, 323), anatomists (322), and garment workers (328). Small excesses of Hodgkin's disease (327) and prostate (320), skin (319), kidney (319), connective tissue (328), and digestive system (319, 320, 324) cancers have been reported from individual studies, but they have not been confirmed by other investigations. Formaldehyde is rapidly metabolized and cleared from plasma; thus, the hypothesis that it causes cancer at sites distant from the point of absorption does not have strong biological plausibility (302).

At present, the epidemiologic data on the human carcinogenicity of formaldehyde are variable, and definitive conclusions cannot be reached. Formaldehyde exposure of subjects was not directly assessed in any of the studies; use of indirect measures may introduce random misclassification and reduce risk estimates towards unity, regardless of study design. Most of the cohort studies are limited by short duration of follow-up and by inadequate statistical power because of small sample sizes and small numbers of deaths. The case-control approach is appropriate for evaluating causes of a rare disease, such as nasal cancer. However, accurate retrospective documentation of exposure may be difficult. The proportional mortality method, used by Walrath and Fraumeni (319), Walrath (320), and Marsh (324), has inherent methodologic limitations (337); apparent excesses in one cause of death may be due to deficits in another. Further epidemiologic studies of the relationship between residential formaldehyde exposure and cancer should be undertaken.

Because the epidemiologic data are preliminary and inconsistent, risk assessment procedures have been used to describe the hazards of formaldehyde exposure. The Risk Estimation Panel of the Consensus Workshop on Formaldehyde (302) recommended that the epidemiologic data not be used for risk estimation and concluded that the animal data on nasal cancers were satisfactory for this purpose. Risk estimation requires the selection of a model to extrapolate from observed effects at high doses to the lower doses anticipated from human exposures at which effects cannot be directly identified. The choice of a particular risk assessment model may have a profound influence on the apparent risk of formaldehyde. The widely contrasting models and risk assessments recently published by the Chemical Industry Institute of Toxicology (338, 339) and by the Consumer Product Safety Commission (340) are illustrative.

The Risk Estimation Panel of the Consensus Workshop (302) proposed that

formaldehyde should not be considered to have a threshold for cancer induction, but it did not strongly support any particular risk estimation model. Panel members agreed that the animal data from the Chemical Industry Institute of Toxicology Study were best described by a nonlinear model, but they were not able to specify the most appropriate nonlinear model. Different nonlinear models may produce differing estimates of risk at low doses, however. The panel considered that a simple linear model would provide a conservative upper bound for the risk at lower doses. Further research may provide new insight concerning the most suitable model and reduce the uncertainties concerning the human carcinogenicity of formaldehyde.

Nonmalignant Respiratory Effects. On the basis of studies of occupationally and domestically exposed populations, formaldehyde has been reported to cause excess respiratory symptoms, acute and chronic reductions of lung function level, and asthma. However, the evidence from these investigations is inconclusive.

Questionnaire surveys of symptoms have been performed on populations selected because of complaints about formaldehyde exposure at home or at work (table 10). These surveys show seemingly high prevalences of respiratory and nonrespiratory symptoms. The investigations of complaints in Washington, Wisconsin, Connecticut, and Texas cannot be readily interpreted because comparison populations were not evaluated and bias may have resulted from the selection of complaining subjects. The studies provide documentation, however, that formaldehyde exposure may occur in the domestic environment.

Thun and coworkers (313) used a more informative and less biased design in a study of 1,396 residents of homes insulated with UFFI and 1,395 residents of homes without UFFI. Subjects were selected from a roster of households insulated with UFFI rather than on the basis of symptom status. By telephone interview the investigators ascertained symptom prevalence over the previous year and the timing of symptom onset in relation to installation of UFFI. The prevalences of wheezing and burning skin were significantly higher in residents of homes with UFFI. Subjects who reported that odor had persisted for greater than 7 days after UFFI installation had the highest incidence of symptoms. The lack of formaldehyde measurements in the home, the low response rate, and the retrospective ascertainment of symptoms detract from the findings. However, this study represents one of the few population-based investigations of residential formaldehyde exposure in which study subjects were selected because of their potential for exposure rather than because of perceived health effects.

Norman and coworkers (341) examined the relationship of residence in a home insulated with UFFI to pulmonary function and respiratory symptoms in school children. Using data gathered during a previous study in Canada, the investigators identified children who had been living in homes with UFFI. Two children from homes without UFFI were matched to each exposed child ($n = 29$) on the basis of 9 variables that had been shown to predict pulmonary function. No association was found between exposure to UFFI and respiratory function or symptoms. Measurements of formaldehyde were not obtained.

Two studies have evaluated the effects of formaldehyde exposure in mobile homes used as offices (314, 315). Both studies included assessment of control populations and measurements of formaldehyde. Their findings were similar and demonstrated an excess of respiratory and nonrespiratory symptoms. Main and Hogan (315) also evaluated pulmonary function and found no effects of formaldehyde exposure; however, the study group included only 39 subjects.

Effects of formaldehyde on respiratory symptoms and pulmonary function have also been assessed cross-sectionally in occupational settings. Alexandersson and colleagues (342) studied 47 workers exposed and 20 workers not exposed to formaldehyde in a carpentry shop. Symptoms were ascertained by questionnaire, and spirometry was performed twice, once on Monday morning and again on Monday afternoon. The exposed workers reported "chest oppression" and symptoms of the eyes, nose, and throat significantly more often than the control subjects. The FEV_1, the FEV_1/FVC ratio, and the MMEF were normal on Monday morning but showed significant, although small, reductions over the working day in the exposed workers. These decrements, which occurred in both smokers and nonsmokers, were not related to the workers' personal formaldehyde exposures.

Levine and coworkers (343) surveyed symptoms and lung function in 90 morticians attending a continuing education course. Levels of spirometric parameters were not reduced in comparison to standard reference populations. When classified by extent of exposure, as estimated by the number of embalmings performed, the high and low exposure subjects did not differ on symptom prevalence or lung function level. Formaldehyde exposure was not measured directly, however.

Studies of workers exposed to phenol formaldehyde have also been considered to provide evidence on the effects of formaldehyde. Two studies with conflicting results have been published (344, 345). Industrial hygiene measurements documented the presence of other contaminants in these workplaces, and, thus, the results of these studies cannot be considered as relevant for domestic formaldehyde exposure.

Thus, current evidence for chronic effects of formaldehyde on lung function derives solely from several small cross-sectional studies. In a cross-sectional study, particularly for a highly irritating exposure such as formaldehyde, the most susceptible segment of the population is likely to be underrepresented. Further, the study populations were too small to detect any but large effects.

Formaldehyde has been reported to be a cause of occupational asthma (301, 346–348), although the mechanism of action is uncertain. Formaldehyde might cause asthma by specific immunologic sensitization or by induction of bronchoconstriction through nonspecific irritation (349); the relative importance of these 2 mechanisms has not been established. Medical (350, 351) and nonmedical publications have raised the concern that concentrations of formaldehyde found in residences may also be associated with asthma. Studies of persons exposed to formaldehyde in their homes have documented complaints of wheezing, chest tightness, and other symptoms compatible with asthma (table 10). However, cases of asthma resulting from domestic exposure to formaldehyde have not been published. In a unique documented case of a woman who developed asthma after installation of UFFI, the offending agent was found to be UFFI dust rather than formaldehyde (352).

Recent case series provide evidence on the rôle of formaldehyde as a cause of asthma at varying concentrations in the domestic and work environments. In these studies, subjects referred to a clinical facility for investigation of suspected formaldehyde-induced asthma were evaluated with bronchial provocation tests. Nordman and colleagues (353) reported that 12 of 230 workers referred to the Institute of Occupational Health

in Finland had positive bronchial provocation tests when exposed to formaldehyde at 2 ppm. In England, Burge and coworkers (354) described 15 workers evaluated for occupational asthma. The investigators concluded that 3 subjects showed specific hypersensitivity to formaldehyde, 2 were affected through irritant mechanisms, and the remaining 10 subjects were probably affected by other agents.

Frigas and associates (355) evaluated 13 subjects referred for evaluation of possible asthma secondary to formaldehyde exposure in the work or home environment. As none of the 13 subjects responded to formaldehyde challenge, the investigators questioned the importance of formaldehyde as a cause of asthma at levels below 3 ppm, the range generally encountered in the domestic environment. However, because this series comprised only 13 subjects, firm conclusions on the role of formaldehyde cannot be drawn from its results. The findings of Nordman and colleagues (353) imply that at most one of the 13 subjects studied by Frigas and associates (355) would be expected to have formaldehyde-induced asthma.

These studies suggest that asthma may be mistakenly attributed to formaldehyde exposure and that the incidence of formaldehyde-induced asthma may be low. Although not widely available, specific bronchial provocation testing with formaldehyde is essential for diagnosis; the clinical history, while important for raising the initial concern about formaldehyde-related asthma, may be misleading.

Neuropsychologic and Behavioral Effects. Questionnaire surveys of symptoms in subjects concerned about formaldehyde exposure in their homes have documented a high prevalence of neuropsychologic symptoms, including headache, memory lapse, fatigue, and difficulty sleeping (table 10). The findings of more rigorous studies that included control populations have been similar.

Olsen and Dossing (314) compared the prevalence of symptoms among workers in mobile home day care centers with the prevalence among workers in 3 permanent structures. The workers in the mobile homes reported significantly more complaints of headache and unnatural fatigue, but memory and concentration did not differ in the 2 groups. Although acknowledging that the workers in the mobile homes were specifically concerned about their exposure to formaldehyde, the investigators concluded that biased reporting of symptoms was an unlikely explanation of their results. They based this conclusion on similar prevalences of symptoms unrelated to formaldehyde in the 2 groups.

In another cross-sectional study, Kilburn and coworkers (356) ascertained the frequency of neurobehavioral, mucous membrane, and respiratory symptoms among 76 histology technicians exposed to formaldehyde but also to xylene and toluene. In comparison with 56 secretaries and clerks, the histology technicians were more likely to experience disturbances of memory, sleep, balance, mood, concentration, and appetite. They were also more likely to report eye irritation, a reduced sense of smell, mucous membrane dryness and irritation, chest tightness, cough, shortness of breath, and palpitations. Each technician estimated the average number of hours per day of exposure to formaldehyde. The prevalence of most symptoms increased with lengthening exposure. Of 44 technicians who completed a 20-item depression scale, only 4 had scores suggesting depression. This study was initiated after discussions with histology technicians who were concerned about exposure to formaldehyde and solvents. Consequently, biased reporting of symptoms must be considered when interpreting the results of this study.

In order to measure neuropsychologic symptoms objectively, Schenker and associates (357) used standardized neuropsychologic tests in a study of 24 residents of 6 homes insulated with UFFI. Nine of 23 subjects reported neuropsychologic symptoms, including memory difficulty, headaches, difficulty concentrating, and emotional lability. Complaints of memory loss were not validated by formal tests. However, 11 of the 14 tested subjects demonstrated a deficit in their attention, and 9 of those 11 also had elevated depression scores. While use of objective tests of neuropsychologic function represents an improvement over questionnaire assessment of symptoms alone, the results of this study are, nonetheless, limited by the lack of a comparison population and the small number of study subjects.

The results of the complaint investigations indicate the need for a careful assessment of the neuropsychologic effects of formaldehyde exposure. The cross-sectional epidemiologic studies that have been undertaken involved small numbers of subjects, and their results are not definitive. Further laboratory investigation is needed to establish biological mechanisms that may underlie the neuropsychologic effects of formaldehyde. Formaldehyde might exert a direct toxic effect on the central nervous system. Alternatively, its odor could make those in contact with formaldehyde more aware of symptoms and more likely to attribute significance to them (302). The development and application of objective neuropsychologic tests to a population-based study group will be essential in clarifying the mechanism of formaldehyde's action.

Summary. Although the irritant properties of formaldehyde are documented, evidence on health effects at concentrations found in residences and offices is inconclusive. Respiratory effects and neurobehavioral impairment have been associated with formaldehyde exposure, but many of the studies may have been biased by the approaches used for subject selection and data collection. These health outcomes should receive further investigation in populations with measured exposure to formaldehyde that have been selected without bias. Appropriate control populations should be included in cross-sectional and cohort studies. Continued investigation of workers exposed to formaldehyde is needed to resolve the current controversy concerning carcinogenicity.

Clinically, formaldehyde should be considered as a potential cause of vague respiratory and neuropsychological symptoms and of asthma, but the diagnosis of formaldehyde-induced asthma should not be made without confirmation by inhalation challenge. Formaldehyde exposure may cause mucous membrane irritation in residents of mobile homes, new homes, and homes with potentially strong sources, such as new carpeting.

Volatile Organic Compounds

Volatile organic compounds (VOC) make up a large and diverse group of organic substances that share the property of volatilizing into the atmosphere at normal room temperatures. Formaldehyde, the VOC of greatest public and regulatory concern, has been extensively investigated in the past 10 yr (see previous section). However, hundreds of other VOC have been detected in indoor air (358, 359). Numerous sources of VOC exist in both residences and office buildings, including paints, adhesives, cleansers, cosmetics, building materials, furnishings,

dry-cleaned clothes, cigarettes, gasoline, printed material, and other consumer products (359). Several studies describing sources and concentrations of VOC have now been completed (table 3).

Wallace and coworkers (359) recently summarized data from 9 studies collected in more than 1,000 homes in the United States and Europe. Although objectives and methodologies differed among the studies, all showed that concentrations of most organic compounds varied widely among homes and were substantially higher indoors than outdoors. The median indoor/outdoor ratio generally ranged between 2 and 5 for different compounds, but was as high as 10 for some compounds in some homes. In the most comprehensive investigation to date, investigators from the Environmental Protection Agency measured 12-h integrated exposures and breath levels of selected VOC in residents of 650 households in 6 communities throughout the United States (33, 359). This study included residents of Bayonne and Elizabeth, New Jersey, communities with petrochemical plants. Even in these communities, which have strong outdoor sources of VOC, levels of most halogenated and nonhalogenated compounds were 5 to 10 times higher indoors than outdoors.

While these studies document widespread exposure to VOC and emphasize the importance of indoor sources, they also demonstrate the difficulty in characterizing personal exposures to a complex mixture of compounds and in apportioning concentrations of specific compounds to specific sources. For example, in another Environmental Protection Agency study, VOC were monitored at 1 outdoor site and at 5 indoor locations in a home for the elderly. More than 350 different VOC were detected, 50 of which were common to all indoor locations. However, another 25 to 50 compounds were unique to each individual location (360). Lebret and colleagues (361) measured week-average concentrations of 45 VOC every other week in 4 homes for 26 wk. They reported that concentrations of some compounds were fairly stable over time, whereas others fluctuated widely. Gammage and coworkers (362) continuously monitored 40 homes in eastern Tennessee. They documented high peak concentrations of certain VOC after application of polishes, waxes, and cleaners. These peak concentrations decayed rapidly in most, but not in all, homes.

In all exposure assessment studies performed to date, concentrations of individual compounds have been an order of magnitude below the maximal permissible levels established for industrial environments. However, concern has been raised about the potential of VOC, even at low concentrations, to cause both acute and chronic effects. Some of the most commonly measured VOC are established or suspected mutagens and/or carcinogens. Additionally, many VOC are mucous membrane irritants, and VOC have been implicated as a cause of building-related illness (see section on building-related illness). Further, synergistic interactions among compounds may result in greater health impact than would be anticipated from simple additivity of effects.

Several experimental studies of the acute effects of VOC have been undertaken in the context of studying building-related illness (363, 364). Interpretation of these studies is limited by the nonspecificity of both the exposure and the symptoms, as well as by weaknesses in the study designs. Specific VOC responsible for health effects have not been isolated, and the nature of the relationship between VOC exposure and building-related illness remains unclear. However, further experimental investigations in which subjects are exposed to controlled concentrations of a single VOC and VOC in various combinations should increase our understanding of the relationship between VOC exposure and acute effects. Ultimately such studies, complemented by epidemiologic studies using comprehensive sampling strategies, can be expected to provide guidance on the need for regulations concerned with building design, building materials, and consumer products.

In contrast, epidemiologic studies of the chronic health effects of VOC are likely to prove extremely difficult. Adequate characterization of personal exposures is not currently feasible for a study of sufficient size and length to detect chronic health effects such as cancer. Consequently, assessment of the risk from chronic exposure to varying concentrations of VOC cannot depend on epidemiologic evidence. Further exposure assessment studies are needed to more fully describe concentrations and sources of VOC, to determine the most common VOC present, and to estimate the range and distribution of exposures in the general population. This information, in combination with toxicologic and experimental data, can provide estimates of risk for the formulation of environmental policies.

Building-related Illness

Introduction. Since the early 1970s, numerous outbreaks of work-related health problems have been described among employees in offices not directly contaminated by industrial processes. Two broad categories of episodes can be distinguished: those characterized by a generally uniform clinical picture for which a specific etiology can often be identified, and those in which affected workers report nonspecific symptoms temporally related to work. Symptoms reported in the latter outbreaks have typically included mucous membrane and eye irritation, cough, chest tightness, fatigue, headache, and malaise. In outbreaks with an identified etiology, a wide spectrum of causative factors has been implicated: immunologic sensitizing agents, infectious agents, specific air contaminants, and environmental conditions, such as temperature and humidity (248, 365). Outbreaks without an identifiable etiology have frequently occurred in new hermetically sealed office buildings and have been called "tight building syndrome" (TBS) or "sick building syndrome."

Terminology for these episodes is not uniform. For the purpose of clarity, we will use the phrase "building-related illness" as an inclusive term to refer to all epidemics of illness occurring in nonindustrial workplaces. We will restrict our use of the term "tight building syndrome" to those epidemics of building-related illness that do not have a specific etiology. We recognize, however, that this term may be somewhat misleading, as some of these epidemics occur in buildings that are not tightly sealed.

Exposures in the Office Environment. New construction techniques and ventilation practices directed at conserving energy have led to increasing problems with air quality in the office environment; the resulting buildup of pollutants is undoubtedly a factor in building-related illness. Many multistory office buildings built since 1965 are constructed with an internal structural support surrounded by a thin continuous outer envelope. The external shell is hung from the central core and usually consists of prefabricated components with sealed windows. This technique is often less expensive than alternatives, and the external shell provides a barrier to uncontrolled infiltration of outside air. Air movement into the modern office building is controlled entirely

through a heating, ventilation, and air-conditioning system that usually cannot be controlled directly for any particular space by its occupants. Frequently, to maximize the extent of usable floor space, the heating, ventilation, and air-conditioning system is located on the rooftop. Such systems tend to be designed to operate over a smaller range of ventilation than systems installed within a structure.

The reduced ventilation rates in modern office buildings may lead not only to a generalized air quality problem but to the development of specific localized problems. The majority of buildings are operated at a positive pressure with regard to outside air. Morris and Wiggin (366) have warned that lowering the static pressure in buildings may actually deprive upper floors of fresh air through a buoyancy or "chimney effect." In some buildings, reduction of operating pressure may result in entry of air from polluted locations, such as underground parking garages, and from exhaust vents placed near the street.

For decades, buildings were constructed with constant air flow volume systems that were designed to heat or cool the space within the building. In newer and more tightly sealed buildings, internal sources, such as lights, machines, and people, may suffice to heat the air. In these buildings, air delivery rates are varied to maintain the temperature and not necessarily to meet air quality needs.

The rapid increase in energy prices in the 1970s led to a reevaluation of ventilation standards and operating practices. As a result, fresh air supplies were frequently reduced to a minimum in office buildings. The American Society for Heating, Refrigerating, and Air Conditioning Engineers revised its recommended ventilation standard for fresh air supply in the absence of smoking (367), recommending 10 cubic feet per minute (cfm) per person. The guidelines recommend between 20 and 30 cfm per person for spaces where smoking is permitted. The economic incentives for reducing the fresh air supply are evident. Depending on local utility prices and climate, the annual cost of supplying and conditioning a cubic foot of air per minute may range from $2.00 to more than $4.00 in most locales. Schools, office buildings, and arenas often require fresh air supply rates greater than 100,000 cfm.

The office environment contains numerous sources of potentially hazardous air pollutants. Cigarette smoking, unvented combustion emissions, and vehicle exhaust may add particles and gases to the air in an office. VOC may be released from adhesive, tiles, vinyl wall coverings, rugs, office furniture, and wet-process copying machines. Solvents, cleansers, pesticides, and fibers may also contaminate the air in an office.

Bacteria and fungi may grow on wet surfaces, air conditioners, ducts, filters, and humidifiers. In the past, disease outbreaks in office workers caused by bacteria and fungi were most common in the winter and possibly were related to recirculation of air. More recent outbreaks of disease have often occurred during the time that coolers are in use; the ventilation system may disseminate microorganisms that proliferate in the drip pans under condensing coils or in the water reservoir of a cooling system. The standing water provides a suitable environment for a variety of microorganisms; the specific organisms that proliferate depend upon available nutrients, the pH of the water, and the temperature of the water. After growth of organisms begins, metabolic products may provide nutrients for other organisms and support a growth chain that includes bacteria, fungi, algae, and amoebae. A slime of viable and dead organisms and spores may develop (368). This material may become aerosolized and distributed by the ventilation system.

Building-related Illness. Outbreaks of illness in office buildings related to some of the specific etiologic agents mentioned above have occurred for many years. However, a new problem, characterized by reports of nonspecific symptoms among building occupants that could not be attributed to specific agents, was first reported in the late 1970s. This problem was soon designated "tight-building syndrome."

TABLE 12

CLASSIFICATION* OF THE ETIOLOGY OF BUILDING-RELATED ILLNESSES IN 356 HEALTH HAZARD EVALUATIONS CONDUCTED BY THE NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH THROUGH DECEMBER 1985

Etiology	(n)	(%)
Inside contaminants	67	19
Outside contaminants	38	11
Contaminants from building materials	14	4
Biological contaminants	19	5
Inadequate ventilation	179	50
Unknown	39	11

* See text for definitions. Data provided by Kenneth Wallingford, personal communications.

To date, most of the information on building-related illness derives from health hazard evaluations conducted by federal and state agencies rather than from formal epidemiologic studies. As of December 1985, the National Institute of Occupational Safety and Health (NIOSH) had completed 365 health hazard evaluations of building-related problems (Kenneth Wallingford, personal communications). These evaluations were not conducted according to a standardized protocol until recently, and their classification by etiology was based on a review of written reports (309). Nevertheless, the results of the health hazard evaluations illustrate the heterogeneous etiologies of problems related to office buildings.

Indoor contaminants were cited as the primary cause of building-related illness in 19% of episodes (table 12). This diverse category included all chemical contaminants, such as copying machines, carbonless copy paper, and tobacco smoke, generated by indoor sources. Contaminants from outdoor sources, such as motor vehicle exhaust or dust from construction, may be drawn into a building through intake vents. In 4% of cases, contaminants from building materials and products, such as formaldehyde from new furnishings and fibrous glass from lined ventilation ducts, were considered to be the responsible agents. The biological contaminants were primarily associated with hypersensitivity pneumonitis.

However, in 179 of the 356 investigations, no specific causal agent other than inadequate ventilation could be identified. Because ventilation measurements were performed rarely in the earlier investigations, this categorization was often based on questionnaire data or on exclusion of other causes. In the more recent investigations, ventilation has been evaluated directly by measuring air flow or indirectly by examining building specifications. Standards developed by the American Society for Heating, Refrigerating, and Air Conditioning Engineers for ventilation and thermal environment have been used as a basis for comparison. However, the specific mechanisms through which inadequate ventilation produces tight building syndrome are unclear.

Recent epidemiological investigations have further characterized the dimensions of tight building syndrome. Finnegan and coworkers (369) determined symptom prevalence in workers in 9 of-

TABLE 13

PREVALENCE (%) OF SYMPTOMS IN BRITISH WORKERS IN RELATIONSHIP TO THE AIR SUPPLY IN THEIR OFFICE BUILDINGS*

	Type of Air Supply			
Symptom	Natural Ventilation ($n = 259$)	No Humidification Air Recirculation ($n = 73$)	Humidification No Air Recirculation ($n = 354$)	Humidification Air Recirculation ($n = 477$)
Nasal	5.8	13.7†	22.4‡	17.2‡
Eye	5.8	8.2	28.3‡	17.6‡
Mucous membrane	8.1	17.8†	37.9‡	32.6‡
Tight chest	2.3	1.1	9.6‡	7.8‡
Shortness of breath	1.6	—	4.3	2.9
Wheeze	3.1	—	5.1	4.4
Headache	15.7	37.0‡	34.7‡	39.5‡
Nosebleed	0.5	—	1.4	2.2
Dry skin	5.7	5.5	16.2‡	14.9‡
Rash	1.9	2.7	3.1	2.9
Itchy skin	2.9	2.7	7.4†	7.2†
Lethargy	13.8	45.2‡	49.9‡	52.5‡

* Based on table II in reference 366. Series included 3 buildings with mechanical ventilation, one with no humidification and air recirculation, 2 with humidification and no air recirculation, and 3 with humidification and air recirculation.

† $p < 0.05$ in comparison with workers in the natural ventilation building.

‡ $p < 0.01$ in comparison with workers in the natural ventilation building.

fice buildings, 3 with natural ventilation, and 6 with mechanical ventilation (table 13). The symptoms were ascertained by questionnaire and considered work-related if onset or worsening was related to working in the building. Symptoms compatible with tight building syndrome were significantly more common in workers in each of the mechanically ventilated buildings. Selection bias cannot explain these findings, as 7 of the 9 buildings were chosen by the investigators for study without prior knowledge of building-related problems. The results of this study also illustrate the potential for selection bias in studies of tight building syndrome; symptom rates in workers in the 2 buildings selected because of complaints were higher than rates in the buildings selected by the investigators.

In a subsequent study, this same investigative group surveyed workers in 2 buildings, one naturally ventilated, and the other mechanically ventilated (370). The prevalences of rhinitis, nasal blockage and dry throat, lethargy, and headache were significantly higher in workers from the building with mechanical ventilation. Measurements of temperature, humidity, air velocity, ion concentrations, CO, O_3, and formaldehyde did not differ between the 2 buildings.

The findings from the health hazard evaluations and the cross-sectional epidemiologic studies implicate ventilation as contributing to tight building syndrome. It remains unclear whether the effects of reduced ventilation are mediated directly by alterations in comfort or indirectly by causing the buildup of other pollutants. Monitoring of offices with work forces affected by tight building syndrome has not shown that concentrations of specific pollutants exceed accepted standards (370, 371).

VOC have been considered as one possible cause of tight building syndrome (372) (see section on VOC). With new analytical methods, hundreds of VOC have been found in indoor air (358, 359), usually at concentrations substantially lower than those permitted in the workplace. However, VOC are generally present in complex mixtures, which might produce health effects even though none would be anticipated on the basis of individual VOC concentrations. Further, the irritant properties of VOC make them plausible etiologic agents for many of the symptoms of tight building syndrome. Many of the known VOC could contribute to the eye, mucous membrane, and respiratory tract irritations common in tight building syndrome.

Data from recent experimental investigations lend support to this hypothesized role of VOC. Ahlstrom and colleagues (373) exposed healthy volunteers to 0.82 ppm of formaldehyde in a chamber. Varying percentages of air from a building where workers had been affected by tight building syndrome were added to the chamber. Symptoms of mucous membrane irritation were 4 times more common when the percentage of air from the office building was increased from 10 to 100%.

Molhave and coworkers (363) exposed 62 healthy volunteers, all of whom had previously complained of symptoms typical of tight building syndrome, to a mixture of 22 VOC commonly found in indoor air. Exposure concentrations were zero, 5, and 25 mg/m^3, corresponding to concentrations found in clean air, in air normally present in new houses, and in very contaminated indoor air, respectively. In a double-blind design, each subject was exposed to a concentration of zero mg/m^3 and to a concentration of either 5 or 25 mg/m^3 of mixed VOC. Subjects' perceptions of air quality, odor, and symptoms were assessed by questionnaire. Subjects rated the air quality unacceptable and reported symptoms of nose and throat irritation and inability to concentrate significantly more often when exposed to either 5 or 25 mg/m^3. Additionally, the investigators objectively evaluated the participants' responses to different exposure levels using the digit span test, the graphic continuous performance test, and a trigeminal nerve irritation test (364). Performance on the digit span test, which measures ability to concentrate and short-term memory, was impaired at both exposure levels; other tests were normal.

Interpretation of these reports is limited by their preliminary nature. Little data were provided, and estimates of effect were not presented. Moreover, the study subjects were selected from a pool of 287 subjects who had all experienced "indoor climate symptoms," primarily irritation of the eyes and upper airways. Thus, these subjects may represent a population particularly sensitive to indoor pollutants. Finally, the odor of the exposure would necessarily limit the degree to which the study could be conducted in a double-blind fashion.

The findings of a recent experimental study suggest that both inadequate ventilation and VOC play a role in tight building syndrome. Sterling and Sterling (372) hypothesized that the nonspecific symptom complex characteristic of tight building syndrome is caused by indoor photochemical smog generated by the action of ultraviolet radiation from fluorescent lights on VOC in indoor air. To test this hypothesis the investigators studied the occupants of 2 buildings, one mechanically ventilated and the other naturally ventilated. On the initial symptom survey, the investigators documented a higher prevalence of nonspecific symptoms in workers in the mechanically ventilated building. Subsequently, the employees completed a questionnaire on

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symptoms and environmental quality twice a week during the 10-wk study period.

Without the employees' knowledge, the investigators varied the percentage of fresh air entering the mechanically ventilated building and replaced the lights with standard cool white fluorescent lights. During the period that a greater percentage of fresh air was circulated, the employees reported an improvement in environmental quality, including better air movement, decreased stuffiness, and more comfortable temperatures. Symptoms of eye irritation decreased 6.8% when the ventilation was changed, 8.0% when the lighting was changed, and 31.2% when both were changed simultaneously. Negative perceptions of environmental quality and reports of eye irritation rose to the levels documented at the start of the experiment when the ventilation and lighting were restored to their original state.

Neither epidemiologic nor experimental studies have identified specific etiologic agents for tight building syndrome. While the experimental studies suggest that low concentrations of VOC in sealed buildings lead to the symptoms of irritation found in tight building syndrome, concentrations of VOC have not been measured in buildings with affected and unaffected work forces. Several investigators have also suggested that high stress levels, precipitated by inability to control environmental conditions in a sealed building, poor labor-management relationships, or other human factors may contribute to building-related illness (308, 374). While such psychological factors may contribute to tight building syndrome, their roles have not been addressed in formal studies. Assessment of the role of psychological factors in tight building syndrome will be difficult; these factors are difficult to quantify and likely to change after tight building syndrome occurs.

Summary. Continued health hazard evaluations of episodes of building-related illnesses are needed to establish the dimensions of the problem and to identify specific and remediable causes. Evaluations of new outbreaks would be more informative if standardized methods were developed and adopted for assessment of exposures and health outcomes. However, studies limited to buildings with affected work forces cannot fully elucidate the causes of tight building syndrome. Additional epidemiologic and experimental studies are needed. Experimental studies in which volunteers are exposed to measured amounts of suspect agents, singly and in combinations characteristic of those found in problem buildings, will help narrow the list of possible etiologic agents. Epidemiologic investigations must not only address health outcomes, but they also must include a comprehensive assessment of engineering, air quality, and psychological aspects of the workplace. Both cross-sectional and longitudinal investigations may be informative if combined with a comprehensive environmental characterization.

Clinically, the diagnosis of a building-related illness should be considered in persons with appropriate symptoms and employment in a sealed building. However, criteria for making this diagnosis in an individual patient have not been established.

Radon and Radon Daughters

Introduction. Exposure to radon daughters, the short-lived decay products of radon, places uranium and other underground miners at an increased risk for lung cancer (15, 375–377). While the lung cancer risk incurred by underground miners has been recognized for a century, the hazard posed by environmental radon and radon daughters has only recently been investigated. Because radon daughters are invariably present in indoor air, exposure to them may be a risk factor for lung cancer in smokers and nonsmokers in the general population. In fact, the lung dose from inhaled radon daughters is the highest to any organ from natural background radioactivity (376).

Uranium and radium, a member of the uranium decay series, are present in all rocks and soils, although the concentrations vary widely (376, 378). Radium decays to radon, a noble gas. Because radon is inert, it can diffuse out of the material in which it forms and enter the atmosphere or dissolve in surrounding water. Radon decays with a half-life of 3.82 days into a series of short-lived solid isotopes collectively referred to as radon daughters (378, 379). The series of daughters includes Po-218, Pb-214, Bi-214, and Po-214, with half-lives ranging from less than 1 s to 26.8 min, and terminates with Pb-210, a more stable radionuclide with a half-life of 22 yr. Two of the daughters, Po-214 and Po-218, emit alpha particles during decay.

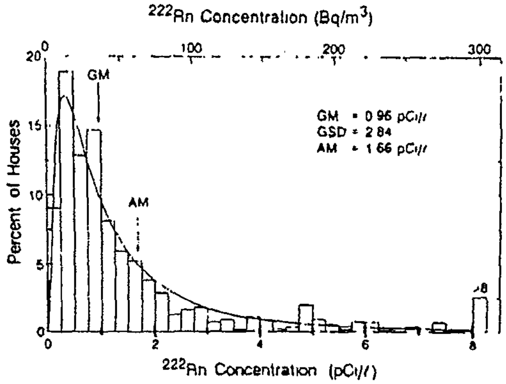
Alpha-decay of inhaled radon daughters while in the respiratory tract is thought to induce the tissue injury that eventually results in malignancy. Although the daughter products release alpha, beta, and gamma energy during their decay, the dose of radioactivity to the lung is due almost exclusively to the alpha particles released by polonium-218 and polonium-214 (377, 380). The alpha particles are presumed to penetrate the epithelial lining of the lung directly and damage the genetic material of the basal cells.

For historical reasons, the concentration of radon daughters is generally expressed as working levels (WL), where 1 WL is any combination of radon daughters in 1 L of air that ultimately releases 1.3×10^5 MeV of alpha energy during decay (379). A concentration of 1 pCi/L of radon translates to about 0.005 WL in a home. Exposure at 1 WL for 170 h equals 1 working level month (WLM) of exposure. The WLM was developed to describe exposure sustained during the average number of hours spent underground by miners. Because most persons spend much more than 170 h in their homes each month, a concentration of 1 WL in a residence results in an exposure much greater than 1 WLM on a monthly basis.

Exposure to Radon. The predominant source of radon in indoor air is the soil beneath structures (376, 381). Radon diffuses through the ground into basements and crawl spaces, and then throughout the air in a home. Other sources include utility natural gas and water used within the home. In homes supplied with water from deep wells in granite rock, radon concentrations may be increased by release of radon that has been dissolved in the water. Short-term variation in the concentration of radon within a home results from changes in air exchange rates, varying meteorologic conditions, and use of water and natural gas.

Radon concentrations have not yet been measured within a large random sample of U.S. homes, although surveys have been undertaken in other countries. Typical radon concentrations range from 0.01 to 4 pCi/L, and much higher levels have been measured in some homes (figure 3). Use of building materials that contain high concentrations of radium and construction of homes on geologic formations composed of granite rich in radium may lead to particularly high levels of radon. Extremely high concentrations of radon have been found in some homes built over the Reading Prong, a geologic formation in eastern Pennsylvania, New Jersey, and Maryland. Some

Fig. 3. Frequency distribution of radon concentrations compiled from selected samples of U.S. homes. Reprinted with permission from reference 379.



conventional homes in Maryland and Pennsylvania have integrated radon concentrations exceeding 20 pCi/L.

Nero (382, 383) examined 35 data sets of radon measurements taken in the United States and identified 22 that provided unbiased data on radon in single family homes. The distribution was similar to that in figure 3, with an average concentration of approximately 1.5 pCi/L; 1 to 3% of the homes exceeded 8 pCi/L. On the basis of this analysis, Nero has suggested that more than 1 million U.S. homes may have annual average radon concentrations exceeding 8 pCi/L.

Lung Cancer and Radon Exposure. Numerous studies of uranium miners and other underground miners have now established a causal association between exposure to radon daughters and lung cancer (375, 384). Animal studies confirm that exposure to radon daughters alone causes lung cancer (377). The human data have come primarily from miners with high exposures to radon daughters, and the risks of lower exposure levels have not yet been well characterized. The shape of the exposure-response relationship between radon daughter exposure and lung cancer risk also has not been established. Other unresolved issues are the lung cancer cell types associated with radon daughter exposure and the nature of the interaction between cigarette smoking and radon daughters.

The relationship between exposure, measured as WLM, and dose to the target tissues in the lung, measured as rads or grays, is extremely complex. Determinants of tissue dose include physical factors, such as the characteristics of the carrier aerosol, the proportions of attached and unattached radon daughters, and the degree of radon daughter equilibrium, as well as biological factors, such as the pattern of respiration, the pattern of particle deposition and clearance, and the locations of the target cells for cancer induction (377, 380). Further, a quality factor for alpha radiation is necessary to convert rads to rems, or grays to sieverts.

These dosimetric considerations suggest that WLM sustained in a mine and in a residence may not yield equivalent tissue doses. Generally, mines are dustier than homes, equilibrium fractions of radon daughters may differ in the 2 environments, the minute ventilation is higher during mining and other underground labor than during normal activities in a home, and the prevalence of respiratory tract abnormalities related to cigarette smoking and other environmental agents may differ in miners and nonminers. Further, exposure to radon daughters in the domestic environment begins at birth, and the anatomy of the child's lung tends to increase dose to the bronchi (377). The effects of these determinants of tissue dose have been examined by computer-modeling techniques. These analyses do not indicate large differences between tissue dose in environmental and occupational settings and support the use of the WLM to describe environmental exposure (377, 385, 386).

To date, epidemiologic investigations of domestic radon daughters as a risk factor for lung cancer have been limited and preliminary. Both descriptive and analytical approaches have been used to examine the association between radon daughter exposure in the home and lung cancer. Techniques for estimating lifetime exposure of persons to radon daughters from indoor air are not yet available, and surrogates based on residence type or on limited measurements have been used in the analytical studies.

In the descriptive studies, incidence or mortality rates for lung cancer within geographic units have been correlated with measures of exposure for inhabitants of these units. Edling and associates (387) compared mortality rates for different Swedish counties with background levels of gamma radiation, which they described as correlated with indoor exposure to radon and its daughters. The correlation coefficients were 0.46 for males and 0.55 for females. Hess and colleagues (388) performed a similar analysis for lung cancer mortality from 1950 to 1969 in the 16 counties of Maine. Using average radon concentrations in water as the measure of exposure, they calculated correlation coefficients of 0.46 for males and 0.65 for females. In a study of 28 Iowa towns served by deep wells, lung cancer incidence increased with the level of radium-226, the source of radon, in the water (389). These descriptive studies, which do not consider the exposures of persons to radon daughters and other agents, can provide only suggestive evidence that radon daughter exposure in the home increases lung cancer risk.

This association has been more directly tested in case-control and cohort studies. Axelson and associates (390) conducted a case-control study with 37 lung cancer cases and 178 control subjects from a rural area of Sweden. Radon daughter exposure was inferred from the characteristics of the subjects' residence at the time of death. Those who lived in stone houses were assumed to be most exposed and those who lived in wooden houses were assumed to be the least exposed, and other types of dwellings were considered to be a source of intermediate exposure. Despite this crude exposure classification, residency in stone houses was associated with a significantly increased odds ratio (age- and sex-adjusted odds ratio = 5.4) in comparison with the reference category of wooden houses. Data concerning cigarette smoking and lifetime residence history were not considered.

In another case-control study in rural Sweden, the study subjects were residents of the island of Oeland, deceased during the period 1960 to 1978 (391). The geologic characteristics of the island were thought to result in strong variation of background radon concentrations within a small area. Inclusion in the study population required at least 30 yr of residence at the same address before death; 23 lung cancer cases and 202 control subjects dead from causes other than lung cancer

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met this criterion. Most of the dwellings were monitored for radon daughters during 3 months of summer and 1 month of winter. The dwellings were also classified on the basis of structural characteristics as in the earlier study of Axelson and associates (390), and cigarette smoking information was obtained from next of kin. Lung cancer risk was significantly associated with radon daughter exposure, as assessed by either the measured concentration or the characteristics of the dwelling, and both crude and smoking-adjusted risk estimates were significantly increased.

Pershagen and coworkers (392) reported the findings of 2 small case-control studies in Sweden on domestic radon daughter exposure; one was drawn from a larger study in northern Sweden, and the other from a twin registry. The investigators assembled each series with 30 case-control pairs divided equally between smokers and nonsmokers. Exposure to radon was estimated from information on dwelling type; the investigators attempted to consider all residences. In the study group from northern Sweden, radon exposures were significantly higher in the smoking cases than in their smoking controls. Estimated exposures were similar in the nonsmoking cases and control subjects in the series from northern Sweden and in the smoking and nonsmoking cases and control subjects in the second series selected from a twin registry.

In the United States, Simpson and Comstock (393) examined the relationship between lung cancer incidence and housing characteristics. During a 12-yr period in Washington County, Maryland, lung cancer incidence in the county's residents was not significantly affected by type of basement construction or building materials. Without specific validation, these dwelling-related variables were assumed to be surrogates for radon daughter exposure.

Because only scant and limited epidemiologic data are available, the hazard posed by radon daughter exposure in indoor air has been addressed primarily through risk estimation procedures. To assess the consequences of exposure by using risk estimation techniques, information on the population distribution of radon daughter exposures in dwellings must be combined in a risk projection model with coefficients that describe the increment in lung cancer occurrence per unit exposure in a risk projection model. For the United States and most other countries, however, the necessary data on radon daughter concentrations are not yet available. The selection of risk coefficients for radon daughter exposure is also problematic. The mining populations that have been studied generally received much higher exposures than arise from the usual environmental sources, and each study has methodologic limitations, particularly with regard to the quality and extent of information on exposure (377, 384, 394, 395). Risk coefficients have also been developed with dosimetric approaches (377, 396).

To perform the risk estimation, a mathematical model must be selected to project the lung cancer cases associated with the exposure to radon daughters. Risk projection models require assumptions concerning the temporal expression of the associated lung cancer cases attributable to radon daughters as well as to the effects of potentially important cofactors, such as age at exposure, age at risk, and cigarette smoking. The 2 most widely applied models are the relative and attributable risk models; the former assumes that the background risk is multiplied by the effects of radon daughter exposure, whereas the latter assumes the addition of the excess risk to background. Thomas and McNeill (394, 395) suggest that the relative risk model is most appropriate for radon daughter exposure and lung cancer.

Estimates of the effect of exposure to environmental radon daughters have been made on the basis of risk models that have varying underlying assumptions (table 14). While the input information for these models may have identified limitations, risk assessment represents the only currently feasible approach for evaluating the extent of the hazard associated with environmental radon and radon daughters. The results of the models indicate that environmental radon daughter exposure poses a substantial risk to the general population. Of approximately 135,000 lung cancer cases annually in the United States, about 10,000 may be attributable to radon daughter exposure; alternatively, about 20% of the lifetime risk of lung cancer in nonsmokers, estimated as 1%, may be explained by radon daughters. These projections, however, are based on average exposures of populations, and individuals may incur much higher risks if they reside in homes with particularly high radon concentrations. New results from studies of miners, in combination with population surveys of radon exposure, should provide more refined risk estimates in the future.

The manner in which radon daughter exposure and cigarette smoking are assumed to interact strongly influences the results of such risk estimation models. If a multiplicative interaction is assumed, then the risks for smokers, already much greater than for nonsmokers, are multiplied by the additional risk from radon daughter exposure. If the interaction between smoking and radon daughter is additive, then the excess risk for smokers is given by the sum of the additional risks incurred by smoking and by radon daughter exposure. The interaction between the

TABLE 14

SELECTED RECENT PROJECTIONS OF THE LUNG CANCER RISK ASSOCIATED WITH ENVIRONMENTAL RADON DAUGHTER EXPOSURE

Author	Risk Projection Model	Findings
Cohen (397)	Attributable risk projection model using various risk coefficients and assuming a mean exposure of 0.22 WLM/yr	Author's best estimate is 10,000 cases/yr
Evans et al. (398)	Attributable risk projection model with lifetime risk coefficient of 10^{-4}/WLM chosen as "most defensible upper bound"	Mean lifetime exposure of 12 WLM gives risk of 0.12%
Steinhausler et al. (399)	Attributable risk projection model with coefficients from 1977 UNSCEAR report; exposures estimated from sampling in Salzburg, Austria	Based on exposure profile, authors' estimate that 15% of lung cancer in Salzburg may be from radon daughters
NCRP Report No. 78 (377)	Combination of dosimetric approach and attributable risk projection model	Lifetime risk for exposure of 0.2 WLM/yr estimated as 0.18%; authors' estimate 9,000 attributable lung cancer deaths annually in U.S.
Thomas et al. (395)	Relative and attributable risk models under varying assumptions; risk coefficients based on literature review and reanalysis of published data	Additional domestic exposure at 0.02 WL from birth causes 2 excess lung cancers per 100

2 agents might take some form other than the purely additive or multiplicative. The presently available epidemiologic evidence indicates an interaction between cigarette smoking and radon daughter exposure that is greater than additive, though the data are not uniformly conclusive (377, 394).

The hypothesis has been advanced that radon daughters directly contribute to the development of lung cancer in both active smokers and in nonsmokers passively exposed to tobacco smoke (400-402). The arguments are complex and will be considered here only for the case of passive smoking. Unquestionably, tobacco smoking increases the concentration of respirable particulates in enclosed spaces. Bergman and coworkers (403, 404) have shown that the introduction of cigarette smoke leads to greater build-up of radon daughters in an unventilated room. Bergman and coworkers interpret this finding as reflecting attachment of daughters to tobacco smoke aerosol, which retards removal by adhesion to room surfaces. Increased exposure to radon daughters would, thus, result from the cigarette smoke. Martell and Sweder (400) and Martell (405) have argued that tobacco smoke increases the concentration of larger particles, which are more likely to be deposited at bronchial bifurcations than the smaller particles present inside uncontaminated, well-ventilated structures.

These speculations require further investigation, and the premises of Martell have been questioned (406). Further, the results of dosimetric modeling indicate that increasing concentrations of particles may decrease the dose received by the basal cells in the tracheobronchial epithelium (377). The dose to these cells falls as the unattached fraction of radon daughters declines. Accordingly, the net effect of tobacco smoke aerosol on the risk of inhaled radon daughters represents the summation of factors tending to increase and decrease dose to target cells. Available data are not sufficient to support a conclusion on the balance of these factors.

TABLE 15
CONTROL MEASURES FOR POLLUTANTS

Pollutant	Control Measures: Equipment and Materials	Control Measures: Ventilation and Design
Respirable particles	High efficiency filters Tight sealing doors and grates Properly drafting chimney Electrostatic precipitators	Zone and ventilate for smoking Supply outside combustion air to heater and fireplace Relocate air intakes Maintain filter system
NO, NO_2	Remove gasoline engine Pilotless ignition	Effective hood vent over source Isolate garage from indoor space
CO	Pilotless ignition Restrict heater use to uninhabited space Use catalytic converter Replace indoor gasoline engines with electric	Supply outside combustion air Vent emission outside Kitchen/hood vent Relocate vents Provide smoking zones Isolate garage from indoor space
CO_2	Check static pressure in return air ducts to make sure return is not overriding fresh air intake	Isolate garage from indoor space
Agents from biological sources	Insulate to prevent condensation Damp-proof foundation, ducts Proper drainage of drip pans under condenser coils Add bacteriocides to steam and water for humidifiers and cooling towers Proper maintenance of filters and ducts Routine cleaning Discard water-damaged floor coverings Do not use cool-mist humidifiers and vaporizers	Maintain inside relative humidities of 35–50% Exhaust bath and kitchen Vent crawl spaces
Formaldehyde	Substitute products such as phenolic resin plywood Seal sources Removal of materials	Increase air exchange to house or office
Radon and radon daughters	Vapor barrier around foundation Damp-proof basement and crawl space Seal cracks and holes in floor traps and drains Install charcoal water-scrubber for well water Completely seal foundation	Vent crawl space Vent sumphole to exterior Subslab depressurization Subslab depressurization Vent bathroom and laundry to exterior
Volatile organic compounds	Substitute products Isolate storage area Apply only according to specifications Do not locate transformers indoors	Use only with adequate ventilation Ventilate laundry, shop Provide separate ventilation to storage area
Asbestos	Removal Injection sealant Wrap pipes with plastic and duct tape	Ventilation does not provide adequate protection

Control of Indoor Air Pollution

Introduction

In this section, we briefly consider the diverse options for achieving acceptable concentrations of air contaminants indoors (table 15). A more comprehensive treatment can be found in the National Research Council's report on indoor air quality (15). Sources may be removed, relocated, or mitigated. Ventilation may be increased to reduce pollutant concentrations throughout a structure or in specific areas. Pollutant concentrations may also be reduced by air cleaning devices, which operate by filtration, adsorption, absorption, electrostatic precipitation, or by other principles. Such devices may be applied to exhaust from the pollutant source, to recirculated air, to the supply air, or to air within the occupied volume.

Source Alteration

While removal of pollutant sources represents the most definitive control method, it is not practicable in many instances. For many sources, evidence of health effects of the released pollutant is not sufficiently compelling; for others, removal may not be feasible. Personal choice may reduce contact with some sources, such as tobacco smoking and woodburning.

Ventilation

In many circumstances, increased air exchange in either a specific zone or throughout a structure effectively reduces pollutant concentrations. Local exhaust of photocopying rooms, areas where smoking is permitted, kitchens, basements, sump-pump areas, and bathrooms reduces pollutant concentrations at relatively low cost. However, zone ventilation is ineffective for emissions that originate from many locations or from materials used throughout a structure. For such widespread sources a localized source of fresh air or alteration of building-use patterns may create zones of adequate air quality. More often, ventilation must be increased throughout the structure.

The extent to which ventilation should be increased is uncertain, although American Society of Heating, Refrigerating, and Air Conditioning Engineers standards provide some guidance. Although 5 cfm per person is twice the ventilation needed to maintain CO_2 concentrations below 0.5%, this organization recommends 7 to 10 cfm for most indoor environments where smoking is not permitted (367). Because of widely varying sensitivity to microorganisms, organic irritants and odorants, and other indoor pollutants, 7 to 10 cfm per person may not provide a satisfactory condition for all occupants. For buildings with occupants affected by a building-related illness, the lower limits of the standards of the American Society of Heating, Refrigerating, and Air Conditioning Engineers may not be sufficient.

Control of ventilation in residences is particularly difficult. Natural ventilation varies with weather conditions, construction, and occupant activities, and air exchange can be readily altered only if the house is equipped with a central heating and cooling system designed to control fresh air intake. Equipping a home with a heat exchanger, either a window unit or a central system, provides a modest increase in air volume exchange.

TABLE 16

COMMERCIALLY AVAILABLE SAMPLING EQUIPMENT FOR INDOOR AIR POLLUTANTS OTHER THAN PARTICULATES

Pollutant Sampler	Manufacturing Company	Sensitivity and Integrating Time	Approximate Cost
Radon: track etch detector	Terradex Corporation 460 N. Wiget Lane Walnut Creek, CA 94598 (415) 938-2545	1 to 3-month exposure 1 to 4 pCi/L	$20 to $60 depending on sensitivity desired
Radon: charcoal canister detector	RTCA 12 West Main Street Elmsford, NY 10523 (914) 347-5010	4 days 0.1 pCi/L	$35/canister includes shipment and analysis costs
Organic vapors	Industrial Scientific Corporation 355 Steubenville Pike Oakdale, PA 15071 (412)758-4353		
Organic vapors: hydrocarbon chemical reaction tubes	National Draeger Inc. P.O. Box 120 Pittsburgh, PA 15230 (412) 787-8383	100 to 3,000 ppm for 4 to 8 h	$3/tube. $900 for pump and accessories
Organic vapors: charcoal badges	3M Corporation Technical Service Department 3M Center St. Paul, MN 55144 (612) 733-1110	Depends on vapors and sampling times; minimum level, 10/mg	$10/badge. $50 to $300 analysis by GC or GC/MS
Formaldehyde: diffusion tube	Air Quality Research, Inc. 901 Grayson Street Berkeley, CA 94710 (415) 644-2097	5 to 7 days	$48 kit, includes 2 monitors analysis and report
Formaldehyde: pro-tek adsorption badge	E.I. Dupont Company Applied Technical Division P.O. Box 110 Kennett Square, PA 19348 1 (800) 344-4900	1.6 to 54 ppm/h up to 7 days or 0.2 to 6.75 ppm/8 h TWA	$20/badge. $25 to $80 for analysis
Formaldehyde diffusion monitor	3M Corporation Technical Service Dept. Building 260-3-2 3M Center St. Paul, MN 55144 (612) 733-1110	0.1 ppm for 8 h	$37/monitor and analysis
NO_2: personal and alarm	MDA Scientific 405 Barclay Blvd Lincolnshire IL 60069 1 (800) 323-2000	2 to 3 ppm; 1/3 TLV electrochemical cell based 15 min to 8 h TWA	$800/dectector $100/output $2.075/ dosimeter. $1.045/ readout unit
NO_2: diffusion tubes	Environmental Sciences and Physiology Harvard School of Public Health 665 Huntington Avenue Boston, MA 02115 (617) 732-1000	500 ppb/h integrated	$10/tube, research only
NO_2: diffusion badge	Environmental Sciences and Physiology Harvard School of Public Health 665 Huntington Avenue Boston, MA 02115 (617) 732-1000	50 ppb/h	$15/badge, research only
CO: passive badge	Lab Safety Supply Co. P.O. Box 1368 Janesville, WI 53547 (608) 754-2345	50 ppm for 8 h produces color change	$3/holder, $12.75/10 indicating papers
CO: detector tube integrated	National Draeger Inc. P.O. Box 120 Pittsburgh, PA 15230 (412) 787-8383	2.5 ppm for 8 h	$255 pump and accessories, $3/tube
CO: detector tube grab	Sensidyne Inc. 12345 Sparkey Road Suite E Largo, FL 33543 (813) 530-3602	5 ppm/min	$130 pump. $2/tube

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TABLE 17

MONITORING EQUIPMENT FOR PARTICULATES* FOR INDOOR AIR QUALITY STUDIES

Instrument Method	Manufacturing Company	Flow Rate or Sensitivity	Approximate Cost
Integrated gravimetric; particles < 3.5 μm diameter	Cyclone separators with filter Several manufacturers cyclones, filters, and pumps	1.7 L/m	Pumps $200 to $700; filters $2; cyclones $20 to $100
Integrated gravimetric; particles between 10 and 3 μm and less than 3 μm diameters	National Bureau of Standards under EPA Contract USEPA Research Triangle Park, NC 27711 (919) 541-2350	6 L/m Separates using filters in series Batteries	Unknown
Integrated gravimetric; particles < 10 μm or < 2.5 μm diameter	Harvard Impactor Environmental Sciences Harvard School of Public Health 665 Huntington Avenue Boston, MA 02115 (617) 732-1000	4 L/m Mass flow controller for 14-day timer, double impactor for sharp cut; fixed location	$2,500
Instantaneous (2/10 s), TSP or RSP; 0.1 to 10 μ forward light-scattering	GCA-Mini-RAM (personal aerosol monitor) GCA Corporation 213 Burlington Road Bedford, MA 01730 (617) 275-5444		
Semiinstantaneous; RSP	Piezobalance (Model 3500) TSI Inc. P.O. Box 64394 St. Paul, MN 55164 (612) 483-0900	>10 μg/m³ 2-min average depending upon concentraton	$3,000 to $5,000
Continuous; RSP submicron light-scattering multisensor monitor	Handheld Aerosol Monitor (HAM) PPM Inc. 11428 Kingston Pike Knoxville, TN 37922 (615) 966-8796	>10 μg/m³ mass concentration, 1.5 L/s	$3,000 to $10,000

* Particles can be measured using a variety of techniques. Using cyclone or impactor separators, smaller size fractions can be collected on filters. Mass can also be measured using the optical properties of particles. For the most part, measuring particles requires equipment costing several hundred to a few thousand dollars. Equipment using filters require that they be preweighted and postweighed in a temperature- and humidity-controlled room.

Air Cleaning

Particles and gases can be cleansed from air with devices manufactured for use in office and residential environments. Devices to remove particles generally operate by mechanical filtration, electrostatic precipitation, or negative ion generation. Depending on the type of device, the efficiency of particle removal depends on particle sizes, filter design, electrostatic effects of the filter medium, and air-flow rate.

Air cleaning devices are frequently used in the home and office settings to control environmental tobacco smoke. The submicron-sized particles in tobacco smoke are not efficiently removed by conventional filters, although more costly high efficiency filters can clear these particles. Thus, most portable residential air cleaners are not satisfactory for tobacco smoke (407, 408). Their filters collect only the coarser particles of a few microns or larger in diameter, and the rated flow capacities of many units are too low to clean the full volume of a room. The most effective units have negative ion generators or high efficiency filters. The negative ions released by a negative ion generator attach to particles and increase particle removal through plateout onto surfaces and through coagulation.

Many of the small air-cleaning devices manufactured for residential use are also unsatisfactory for pollens and other indoor allergens (409). High efficiency filtration devices are more effective for removal, but their clinical utility has not been established (409, 410). Pollen exposure can be reduced by use of a conventional window air conditioner (411, 412). These devices can cool inside air without mixing in outside air (409, 412).

Gases can be removed from indoor air by chemical absorption of reactive substances or by physical adsorption onto surfaces. Activated charcoal filters, silica gel, activated alumina, and alumina oxide impregnated with potassium permanganate have been used for industrial air cleaning for years. More recently, hybrid air cleaners for residential use have included devices for removing gases. Removal efficiency approaching 25% for formaldehyde and 45% for NO_2 has been reported (408). However, the test situation in this report, a 2-h trial in a closed chamber, does not realistically simulate the circumstances of use in a home.

Remedial Action for Radon

The recognition that high levels of radon occur in some homes has led to the development and implementation of procedures for mitigation (376). Entry of radon into a home can be reduced by techniques that direct the radon away from the home, by sealing cracks and other portals of entry, by venting sump pumps, and by removing materials that are high in radium. Air treatment also reduces radon daughter concentrations as does increasing the air exchange rate.

Assessment of Indoor Air Quality

Continuous and integrated samplers are now available for many pollutants in indoor air. The continuous samplers are expensive devices that record real-time concentrations. The instruments for CO, NO_2, formaldehyde, CO_2, and other gases operate on electrochemical, chemoluminescent, or infrared absorbing principles. Costs range between $2,000 for a single gas detection system to more than $12,000 for a tunable wavelength multigas detector. Calibration gases and recording devices add to the costs of using continuous instruments.

Fortunately, less expensive samplers that integrate pollutant concentrations over time have been developed for many gases and particles (38, 39). We list some of these devices in tables 16 and 17, without implying our endorsement. Many of these devices operate by permeation or diffusion; for example, passive samplers are available for NO_2, radon, formaldehyde, and several other organic vapors. Other devices are more sophisticated and incorporate a pump to move air across a filter or vapor trap. With the exception of the colorimetric stain tubes available for industrial hygiene applications, most of these samplers require a laboratory for analysis.

Assessment of particle exposures may require special techniques. For some applications, particle mass alone may be sufficiently informative, but characterization of particle size, morphology, and

chemical and elemental composition may also be necessary.

Conclusions

Research Recommendations

With the exception of airborne infection, the health effects of indoor air pollution received little attention until the early 1970s (413). The research performed subsequently has convincingly demonstrated the importance of indoor environments in determining personal exposures, but it has left unanswered many questions concerning health effects. We will address research needs related to methodology and to specific pollutants.

Methodology. In designing investigations of the health effects of indoor and outdoor pollutants, exposure assessment must be guided by the concept of total personal exposure (figure 1). The optimal approach for exposure assessment is measurement of each subject's personal exposure. For certain pollutants, such as NO_2 and CO, this approach is now feasible. For some pollutants, however, accurate and inexpensive devices are not available, and surrogate measures of exposure, such as source descriptions, are often used in the place of measurements. Studies of personal exposure have shown that misclassification of exposure is inevitably introduced by use of surrogate measures. Therefore, when an investigator relies on surrogate sources of exposure information, the extent of the resulting misclassification should be measured and considered in interpreting the study's results. For certain pollutants biological markers may quantify exposure.

In this review, we have described many studies limited by potential confounding and inadequate sample size. At the concentrations of pollutants generally found in indoor air in U.S. buildings, the anticipated health effects to be evaluated will often be subtle and of small magnitude. Investigations of such low-level effects should not be undertaken without assessment of sample size requirements. Consideration must also be given to misclassification of health outcomes and of exposure and to the implications of misclassification for sample size needs.

Epidemiologic investigations must provide accurate estimates of the low-level effects anticipated for many environmental pollutants. Apparent small effects may be introduced by uncontrolled confounding by environmental and host factors. Therefore, accurate measurement of potentially important covariates must be incorporated into the study design.

Involuntary Exposure to Tobacco Smoke. Research recommendations on involuntary smoking must consider the extensive data available on active smoking, which have long provided sufficient rationale for smoking prevention and cessation. In the face of incontrovertible evidence on active smoking, further research on involuntary smoking is warranted to describe more fully effects on infants and children, to characterize further nonmalignant effects on adults of exposure at home and at work, and to develop more precise risk estimates for lung cancer associated with involuntary smoking. It must be recognized that involuntary smoking affords an important research opportunity for describing exposure-response relationships for a potent and ubiquitous environmental pollutant. Furthermore, the results of research on involuntary smoking are needed to determine the magnitude and acceptability of risks incurred by nonsmokers.

Nitrogen Dioxide. Data from investigations of NO_2 exposure and respiratory illnesses indicate that the magnitude of the effect is likely to be small and less than that of involuntary exposure to tobacco smoke. However, because more than half of U.S. homes have gas cooking stoves and childhood respiratory illness is extremely common, even a small effect of NO_2 is of public health importance. In order to detect associations of the anticipated small magnitude, future investigations should employ direct measurements of exposure, rather than using surrogate variables. Infants and other potentially susceptible groups seem the most suitable populations for study.

Woodsmoke. Woodsmoke is a complex mixture of gases and particles that have a wide range of potential respiratory effects. The unconfirmed observations of Honicky and coworkers (241) that woodsmoke causes acute respiratory illnesses and symptoms in U.S. children require further study. Investigations in less developed countries suggest that domestic smoke exposure contributes to the development of chronic lung disease. This important hypothesis cannot be tested with sufficient sensitivity in most populations in the United States, but should be pursued in appropriate locales.

Formaldehyde. Although the irritant properties of formaldehyde are documented, evidence on health effects at concentrations found in residences and offices is inconclusive. Respiratory effects and neurobehavioral impairment have been associated with formaldehyde exposure, but many of the studies may have been biased by the approaches used for subject selection and data collection. These health outcomes should receive further investigation in populations selected without bias and with measured exposures. Appropriate control populations should be included in cross-sectional and cohort studies. Continued investigation of workers exposed to formaldehyde is needed to resolve the current controversy concerning carcinogenicity.

Radon and Radon Daughters. Radon daughters, like tobacco smoke, are an established cause of lung cancer. Research needs on environmental exposure relate to more precise quantitation of the risks of lung cancer. The requisite information includes population-based data on the distribution of exposure, risk estimates developed at lower levels of exposure than sustained by many of the mining groups evaluated to date, and improved understanding of factors modifying the risks of radon daughter exposure and of the temporal expression of radon-related lung cancer. Epidemiologic investigations of the association between domestic exposure and lung cancer may be informative, but methods for estimating exposures must be further developed.

Building-related Illness. Health hazard evaluations have documented the syndrome of building-related illness, but have not adequately defined its clinical dimensions and causes. Further health hazard evaluations of new outbreaks would be more informative if standardized methods were developed and adopted for assessment of exposures and health outcomes. However, more rigorous epidemiologic methods should also be applied to the problem of building-related illness. Both cross-sectional and longitudinal investigations may be informative if combined with a comprehensive environmental characterization.

Clinical Implications

Although much of the evidence on the health effects of indoor air pollution remains equivocal, some of the exposure-disease associations are established and clinically relevant. Involuntary smoking contributes to lower respiratory illnesses in infants, and mothers should be advised about this adverse effect and the possibility that their smoking will harm their child's developing lung. Active smokers should consider the lung cancer risk that their smoking imposes on nonsmokers.

Carbon monoxide poisoning and hypersensitivity pneumonitis are well described clinical entities that may be overlooked. Indoor routes of exposure should also be considered in outbreaks of Legionnaires' disease, *Aspergillus*, and other infections. High radon and radon daughter levels should prompt mitigation.

The evidence is less compelling for other exposures, and we cannot provide firm guidance. For woodsmoke, the study reported by Honicky and coworkers (241) suggests that woodstoves may cause recurrent respiratory illness in children, but the findings have not been confirmed. The effects of formaldehyde exposure in residences, offices, and other environments also have not been well-characterized. Formaldehyde should be considered as a potential cause of vague respiratory and neuropsychologic symptoms and of asthma, but the diagnosis of formaldehyde-induced asthma should not be made without confirmation by inhalation challenge. Formaldehyde exposure may cause mucous membrane irritation in residents of mobile homes, new homes, and homes with potentially strong sources, such as new carpeting. The diagnosis of a building-related illness should be considered in persons with appropriate symptoms and employment in a sealed building. However, criteria for making this diagnosis in an individual patient have not been established.

Some exposures to indoor air pollutants are probably not associated with adverse effects that are clinically relevant. The epidemiologic studies indicate only minimal effects of NO_2 from gas stoves. Woodsmoke and involuntary exposure to tobacco smoke have not been associated with short-term effects in adults, but the relevant data are scant.

Health care providers can offer some practical suggestions to patients who ask about air cleaning. Only a few of the commercially available devices effectively remove tobacco smoke and pollens (405, 406). However, closing windows and using a window air conditioner reduces pollen counts (407, 409, 410). Some exposures can be readily controlled by removal of the source, such as tobacco smoking and unvented space heaters, or by proper venting and use of exhaust fans, such as with gas stoves. Merely opening a window to increase ventilation may be effective.

Health care providers may also be viewed as expert on the health effects of indoor air pollution, as on other health topics. In recent years, the communications media have regularly transmitted the results of studies and reports on indoor air pollution. Patients may turn to their health care providers for information on radon, involuntary smoking, gas stoves, and other prevalent exposures. This review provides some information for answering these questions.

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Addendum

During 1987, several new sources of information on indoor air pollution and health have been published. The 4th International Conference on Indoor Air Quality and Climate was held in August 1987. The proceedings were published by the Institute for Water, Soil and Air Hygiene in Berlin (mailing address: Institut für Wasser-, Boden- und Lufthygiene des Bundesgesundheitsamtes, Corrensplatz 1, D-1000 Berlin 33). The U.S. Environmental Protection Agency report "EPA Indoor Air Quality Implementation Plan" and its appendices provide a comprehensive review. Two new reports on environmental radon are available: "Lung Cancer Risk from Indoor Exposure to Radon Daughters," Publication 50 of The International Commission on Radiological Protection, and the report of the Biological Effects of Ionizing Radiation (BEIR) IV Alpha Committee of the National Academy of Sciences.

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THE UNIVERSITY OF TULSA
Division of Continuing Education
The Center for Environmental Research and Technology

Radon Control and IAQ Concerns in Underventilated Buildings: School Studies

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EXTENDED HEATING, VENTILATING AND AIR CONDITIONING DIAGNOSTICS IN SCHOOLS IN MAINE

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ABSTRACT

An extensive effort to assess the effects of HVAC system operation on the indoor radon levels was conducted. Many schools in the EPA School Evaluation Program have been found to have disabled or malfunctioning outside air on the ventilation system. The outside air in the Maine schools had been disabled. This condition was corrected using professional HVAC and control contractors. Measurements were made of radon levels, total and outside airflows, pressure differentials across the building shell and sub-slab radon levels. Exhaust ventilation, built up air handlers and unit ventilators were investigated. A heat recovery ventilator was added to a room that had leaky window sash as the outside air supply for a passive roof vent system. The passive vents have been blocked off.

INTRODUCTION

In August, 1990, extended radon diagnostics were performed in two Maine Schools. The purpose was to assess the effects of returning the heating, ventilation and air conditioning (HVAC) system to the original operating specifications would have on indoor radon levels. This effort was part of the 1990 School Evaluation Program[1]. Measurements of radon, air pressure differences across the building shell and carbon dioxide levels[2] were made to help judge the system changes. While a large amount of data was collected, these measurements were open to a number of interpretations because the radon levels found in the schoolrooms during the extended diagnostics week were much lower than were found by the screening measurements made in April, 1990.

In December of 1990, followup measurements were made at the Gray High School and Russell Elementary School in Gray, Maine. The purpose of these measurements was to provide a basis upon which to judge the effect of the HVAC improvements on radon levels, air pressure relationships and carbon dioxide concentrations in occupied rooms. December was a good time to make this assessment because it represented a worst case scenario. That is, the outside air dampers in the unit ventilators and built up air handlers were closed to minimum and the competing stack effect was at the maximum. Both conditions are the result of the low outdoor temperatures found in Maine at that time of year. The measurements were carried out by a team of people. The team included : Gene Fisher and Bob Thompson USEPA Office of Radiation Programs, Washington, D.C. ; Bruce Harris, USEPA, AEERL, Radon Branch, Research Triangle Park, NC; Bill Turner, Fred McKnight, H.L. Turner Group, Harrison, Maine; Terry Brennan, Camroden Associates, Oriskany, New York; and Gene Moreau, Bob Stillwell, Maine Department of Health Engineering, Augusta, Maine.

A special note of thanks is extended to the Maine Department of Health for their active participation in this evaluation.

PROCEDURE

The evaluation consisted of a visual inspection and measurement of key performance related variables in the Gray High School and the Russell Elementary School.

An extensive set of measurements were made in the High School. The following measurements were made :

continuous radon (pulse ionization and semi-conductor)
continuous air pressure differences (variable capacitance)
carbon dioxide survey (infrared spectrometer)

Continuous radon monitors were placed in rooms 2, 7, 17, 31, 32, 33, the Guidance Office and the Conference Room. The monitors used were eight Honeywell continuous radon monitors and two femto-Tech continuous radon monitors (room 33 and room 7). The Honeywell units provide mean radon levels for 4 hour intervals and the femto-Techs for 1 hour intervals. Air pressure differences were monitored across the floor slab in rooms, 33, 7, the Conference Room and the Guidance Office. Variable capacitance chambers manufactured by Setra were connected to a data logger provided by EPA to collect pressure difference data. Calibration curves were made for each sensor using a micromanometer. Ventilation rates, outside air fractions and ventilation effectiveness were estimated by making a survey of carbon dioxide levels in the occupied classrooms. These could then be compared to carbon dioxide measurements made in the same rooms at the end of the previous school year. Data was collected from 12/18/90 until 1/16/91. This afforded the opportunity to see the classrooms operated both normally and with school in recess for the Christmas Vacation.

Additionally, measurements of sub slab radon were made in the High School and the nearby Middle School. A carbon dioxide survey was also made in the Middle School. The Middle School is very close to the High School but does not seem to have nearly the elevated radon levels that the High School does. These measurements were made to determine whether the Middle School radon levels were lower due to lower source term, construction characteristics or HVAC operation and design. The radon levels under both schools were in the range of 2000 to 4000 pCi/L. There is no evidence that the source strength is the variable causing the large difference in the radon levels in the two schools.

RESULTS

Overview Of Results

The results of this investigation can be briefly summarized in a few lines. The evidence supporting these conclusions are then presented.

1) average radon levels that do not distinguish between occupied and unoccupied

conditions can be misleading

2) the operation of the air handlers, both outside air and exhaust only, has a definite reducing effect on the radon concentrations in the rooms

3) the decay rate of the radon after the air handler turns on is less than would be expected given the amount of outside air that is introduced because the radon is still entering due to negative building air pressure

4) repairing the outside air functions of the air handler made dramatic improvements in the carbon dioxide levels in the rooms where outside air was introduced.

5) while effective and reliable at solving radon problems, soil depressurization in rooms with inadequate ventilation leaves children sitting in high concentrations of CO_2 and other indoor air contaminants for which CO_2 levels are an indicator.

<u>Effect Of Outside Air Improvements On Radon Levels And Dynamics</u>

Introduction--

Continuous radon levels were monitored in eight rooms of the High School. Rooms 33 and 7 are going to be used to illustrate the effects of the air handler operation on radon levels in classrooms. The resolution of the femto-Tech units in these rooms allows one hour radon levels to be used in the analysis. These rooms are representative of the two different air handling systems - exhaust fans only and unit ventilators with passive relief. Room 33 is in the new wing of the high school, contains a unit ventilator and has repeatedly shown the highest average radon levels and spikes. Room 7 is in the old wing, which has exhaust only ventilation and has shown high radon levels. The only fan powered outside air that can potentially enter Room 7 is from the gym air handlers, when they are running. Otherwise, outside air to Room 7 consists of whatever is drawn in through leakage in the building shell, window wall and corridor.

The next two major sections will examine first Room 33, the unit ventilator room and then Room 7, the exhaust only room, in detail.

Room 33 - Unit Ventilator Ventilation--

The results of the continuous monitoring in Room 33 are shown in Figure 1. Notice that the "rain spike" in this room on Christmas eve rises from 8 to 90 pCi/L and

drops again to 16 pCi/L in a 24 hour period. This is far more severe than in other monitored rooms, indicating that a substantial amount of radon is available to enter this room. As in Room 7, the radon levels in this room drop quickly when the ventilation turns on. This can be seen at the points labeled "Air Handler On" in Figure 1. Notice that on Christmas eve during a rain storm there is large spike in the radon concentration. This spike is seen in every room monitored and is interpreted as a rain spike.

The dynamics of the drop in radon that occurs when the unit ventilator comes on is illustrated by Figure 2. This graph shows the 24 hour period of December 19, 1991. Between midnight and 6 AM the radon level hovers around 17 pCi/L. At 6 AM when the unit ventilator is turned on by a timeclock control, the radon level drops in an exponential decay until it reaches a minimum of around 2 pCi/L in the late afternoon. An exponential decay of contaminant level is expected when dilution air is introduced into the room. After the unit ventilator is turned off, the radon levels begin to climb until they reach a level of 7 pCi/L again at midnight. The mean radon concentration for this 24 hour period is 8.9 pCi/L and for the occupied time it is 6 pCi/L. However, for the lowest nine hour period the mean radon level is 3.8 pCi/L. This means that the dose delivered to the occupants could be reduced 37% by starting the unit ventilator three hours earlier.

NOTE : A correction for built up radon decay products in the continuous monitor is not required for the pulse ionization device used because the decay products are collected using an electric field without being counted. However, due to diffusion lag into and outof the sensitive volume, a one hour time delay is observed in the radon dynamic.

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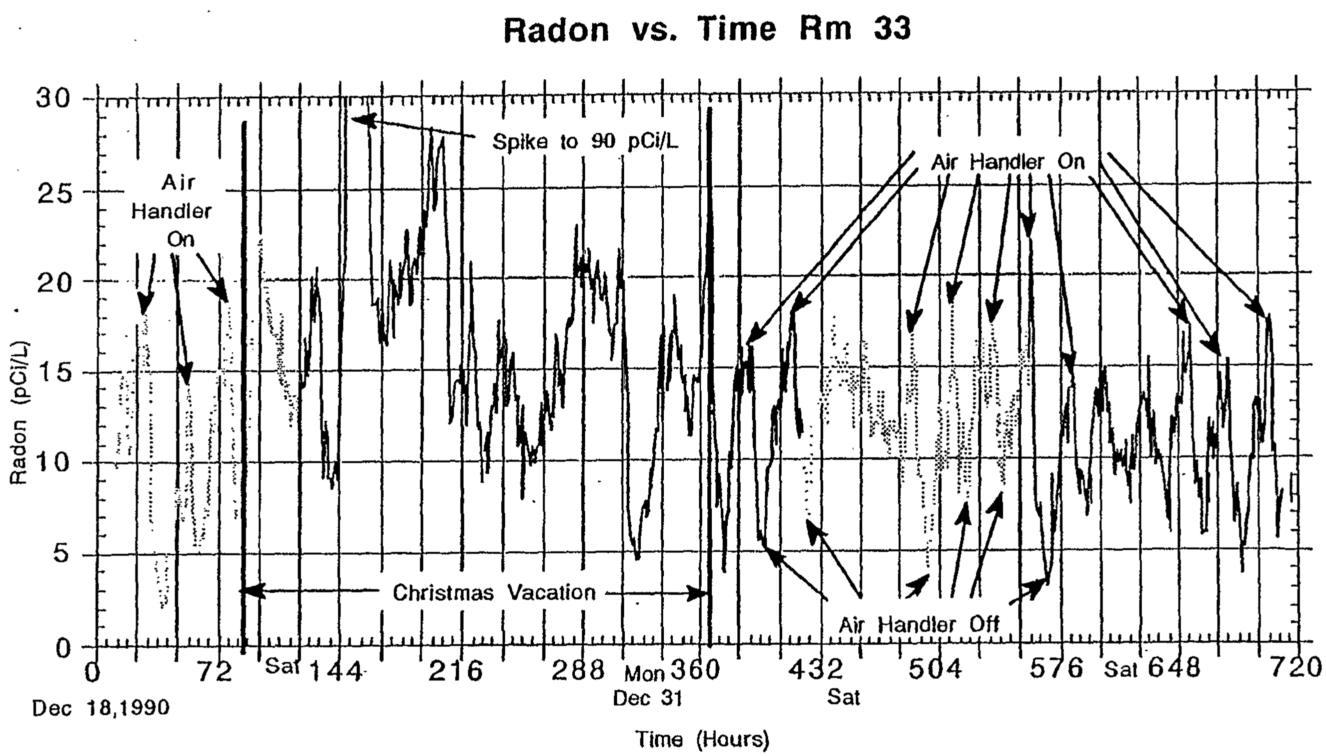
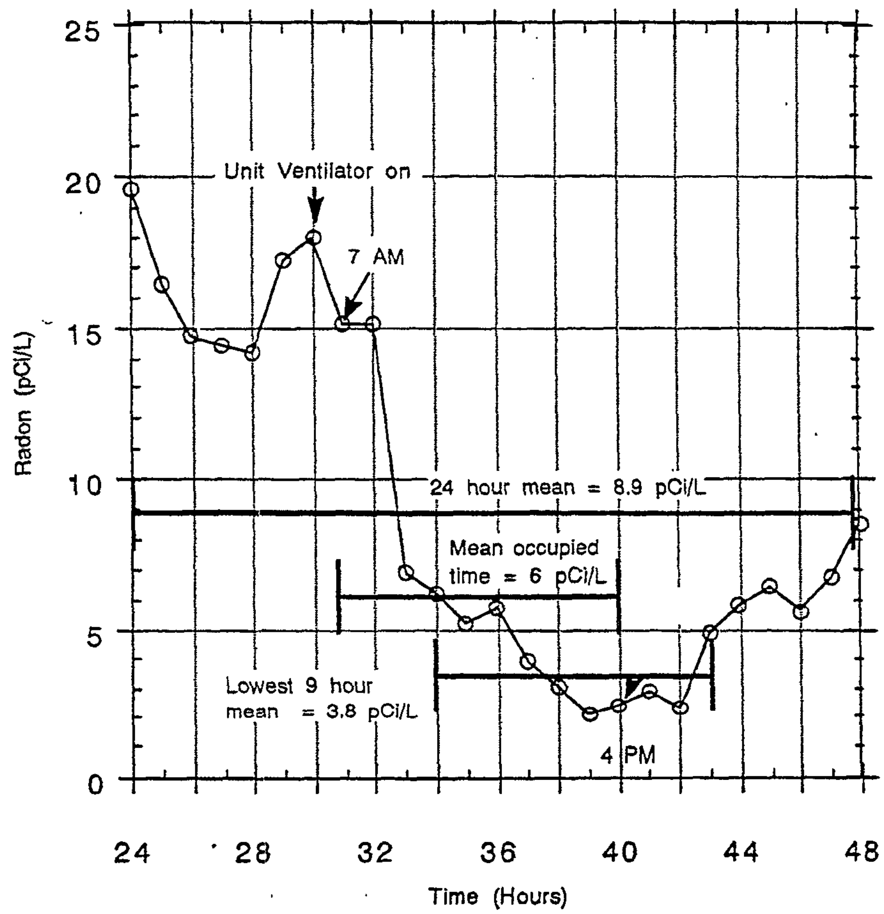


Figure 1 - Radon Levels in Room 33

BG

Figure 2 - Radon Dynamics in Unit Ventilator Room 33



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While for this one day, the 19th of December the mean radon level for the occupied time period was 6 pCi/L, it was not so for other occupied days. In fact, the average occupied time radon level for the entire monitored period shown in Figure 1 is a higher 7.8 pCi/L. This is still 28% lower than the 10.8 pCi/L mean for the entire time period.

Another approach to understanding this dynamic is to apply tracer decay theory. This has been done in the analysis shown in Figure 3. Figure 3 was created by taking the decay curves for all the occupied days during the monitoring period and plotting them on a single graph. The time scale has been changed from consecutive hours to hours after the unit ventilator turns on. The result is a scattergram that plots all the decay data for all the occupied days on top of each other.

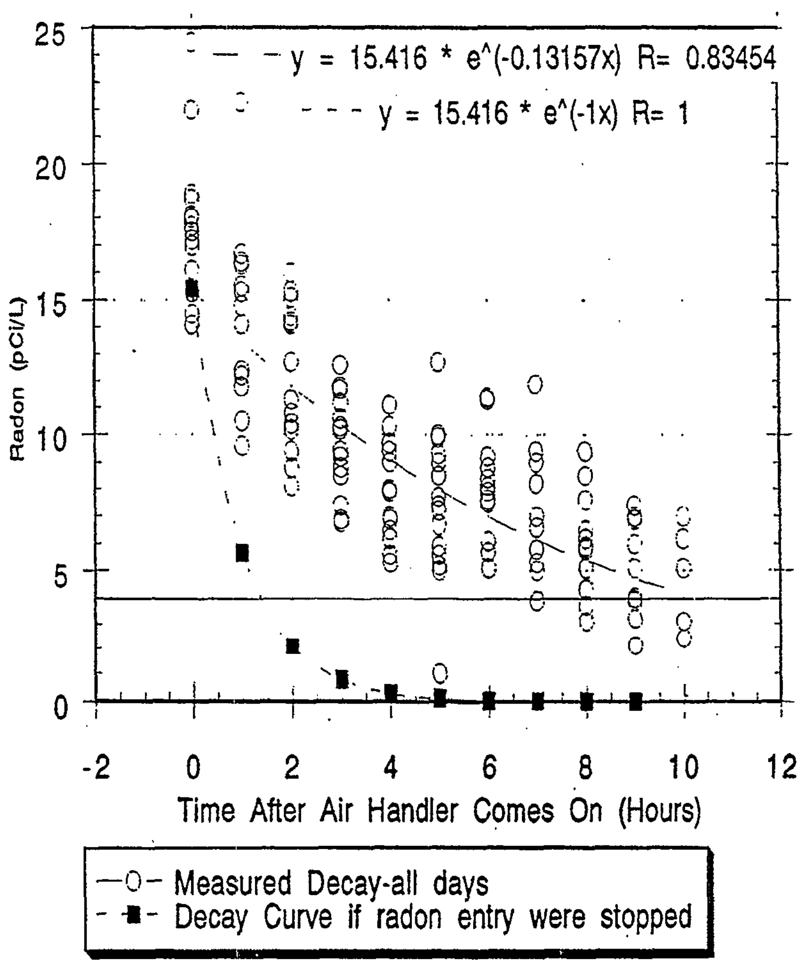
If a given amount of contaminant is released into a room and then allowed to be removed by dilution with ventilation air, it is expected that the concentration of the contaminant will decay exponentially with time[3]. The rate at which it decays is described by the solution to the continuity equation. This is given as the following :

1) $$C(t) = C(0) \times e^{Nt}$$

where : $C(t)$ = concentration at time t
$C(0)$ = concentration at the start of the decay
N = airchange rate in air changes per hour
t = time in hours

By fitting an exponential decay curve to the data in Figure 3, the decay rate and the air exchange rate for the average day during this monitoring period can be determined. It is obvious from this curve that if the radon level at the start of the day is greater than about 8 pCi/L, the mean level during the day would not get below 4 pCi/L. The curve fit yields an air exchange rate of 0.13 air changes per hour (ACH). By direct measurement of outside air, it is known that the air exchange rate in the room is 1 ACH. This discrepancy is explained in the following way. In order for equation 1) to describe radon concentrations, the entry rate of radon after the start of the decay must be zero. The introduction of outside air has not stopped radon from entering the room. This is easily verified by a glance at the air pressure difference between the room air and the sub slab air. The room air was at a lower pressure than the sub slab air during the entire monitoring period. When the unit ventilator turned on, this difference became smaller, but the room was still negative relative to the sub slab. The radon

Figure 3 - Reduction Rate of Radon in Room 33
Air Handler On - All Days Combined



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entry rate may have been reduced but it certainly was not stopped. If the room was pressurized by the unit ventilator then the radon concentration would have dropped according to the lower curve in Figure 3. The radon concentration would be below 4 pCi/L in a matter of an hour.

In fact, it is likely that this is the case in this room during the spring and fall when the outside temperature is warmer than in January. This is expected for two reasons. One, warmer outside air means a reduction in the air pressure differences induced by the stack effect. Two, when the outside air is warm enough gains from body heat will overheat the room and cause the outside air dampers to open more. This will increase the outside air volume and contribute to pressurizing the room.

Lastly, the room could potentially be pressurized even under the worst case condition represented by these test results. This could be accomplished by air sealing the room so that the minimum outside air flow rate would pressurize the room. Not only would this control the indoor radon but it also would result in energy savings by reducing air infiltration.

Room 7 - Exhaust Only Ventilation--

Figure 4 shows the continuous radon data in Room 7. The data begins on December 18, 1990. Christmas vacation began on December 20, 1990 and ended January 2, 1991. The radon levels in this room plummet whenever the rooftop exhaust fans turn on (see the points labeled "Air Handlers On" in Figure 1). This effect is repeatable. The radon levels drop in spite of the fact that operation of the exhaust fans drives the air pressure difference between room 7 air and the sub slab air 3 pascals lower. It is likely that the amount of radon entering the room increases when the fans turn on. Although more soil air is being drawn in by the operation of the fans, the dilution effect of the increased ventilation from above grade overwhelms the increased radon entry. Unfortunately, the increased entry is not overwhelmed enough so that the occupied radon levels are below 4 pCi/L, but are instead 7.1 pCi/L.

Figure 5 shows the agglomerated radon data for the occupied days in Room 7. This graph was generated in the same way that Figure 3 was for Room 33. The general trend of decreasing radon levels after the exhaust fan turns on is obvious. There is a great deal more scatter in this data than there was in the data from Room 33 (the unit ventilator room). The curve fit to this data shows an effective ventilation rate of only 0.065 ACH, while the measured exhaust rate informs us that there is actually 0.63 ACH (shown as the theoretical curve in Figure 5). The data from Figure 3 and Figure 5 are combined in a single graph in Figure 6. This figure highlights the similarities and differences between the dynamics of the two rooms. Notice that the theoretical curves for the two rooms almost coincide, even though the fan powered air exchange rates

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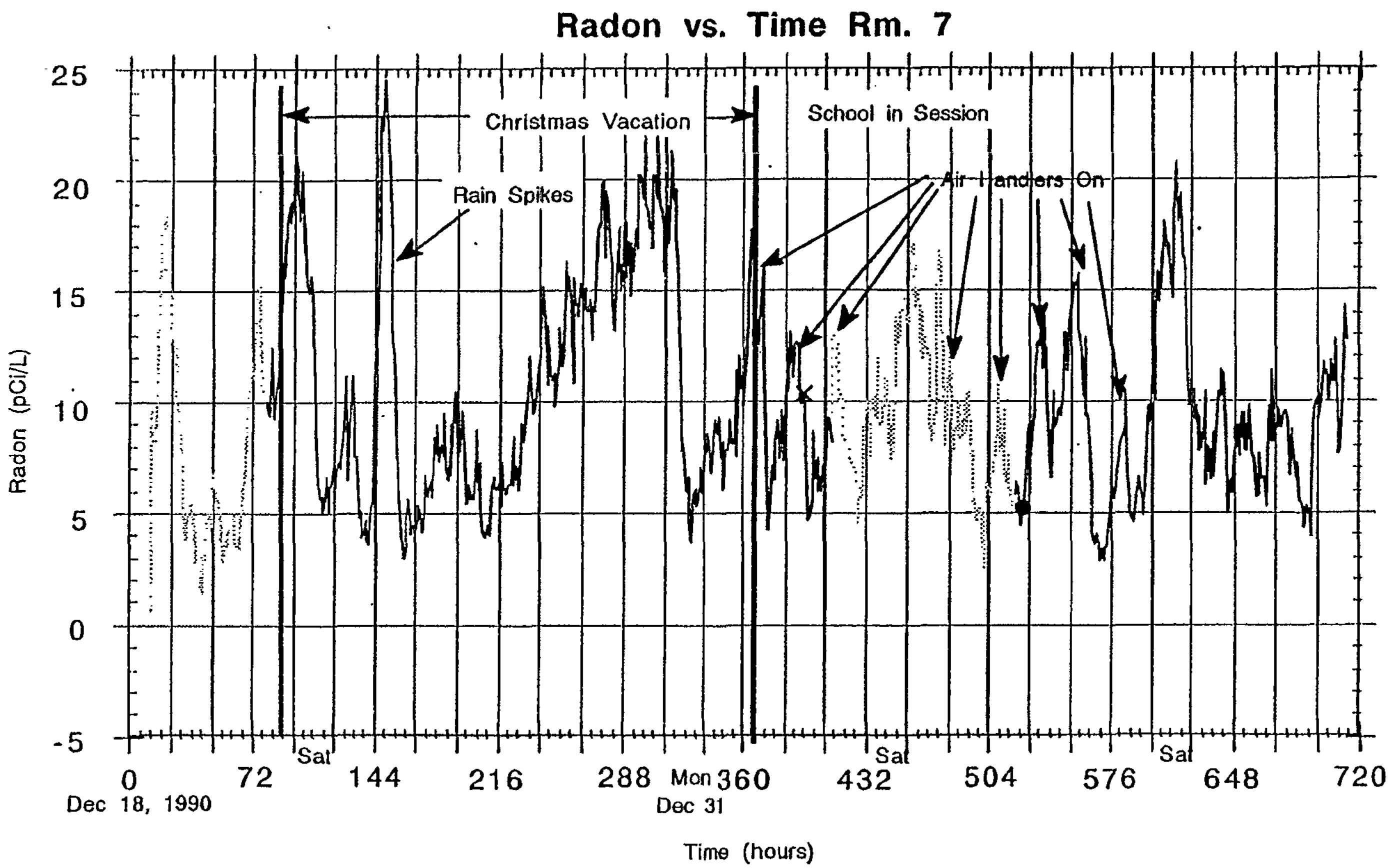

Figure 4 - Radon Levels in Room 7

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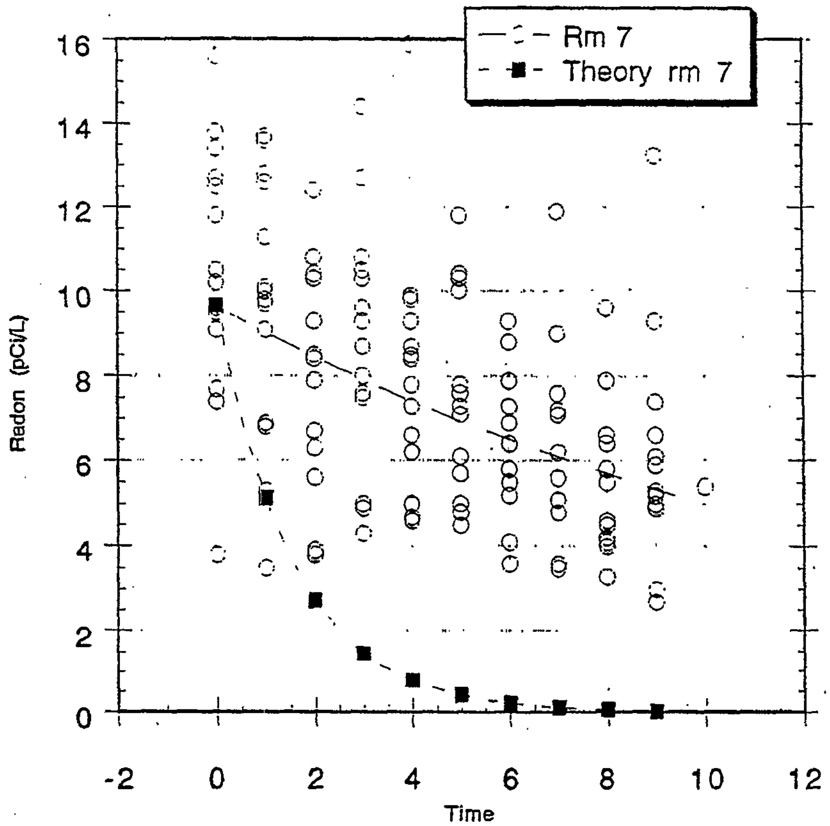
Figure 5 -Reduction Rate of Radon in Room 7
ExhaustFanOn-AllDays



— — y = 9.6569 * e^(-0.065616x) R= 0.4982

- - - y = 9.67 * e^(-0.63x) R= 1

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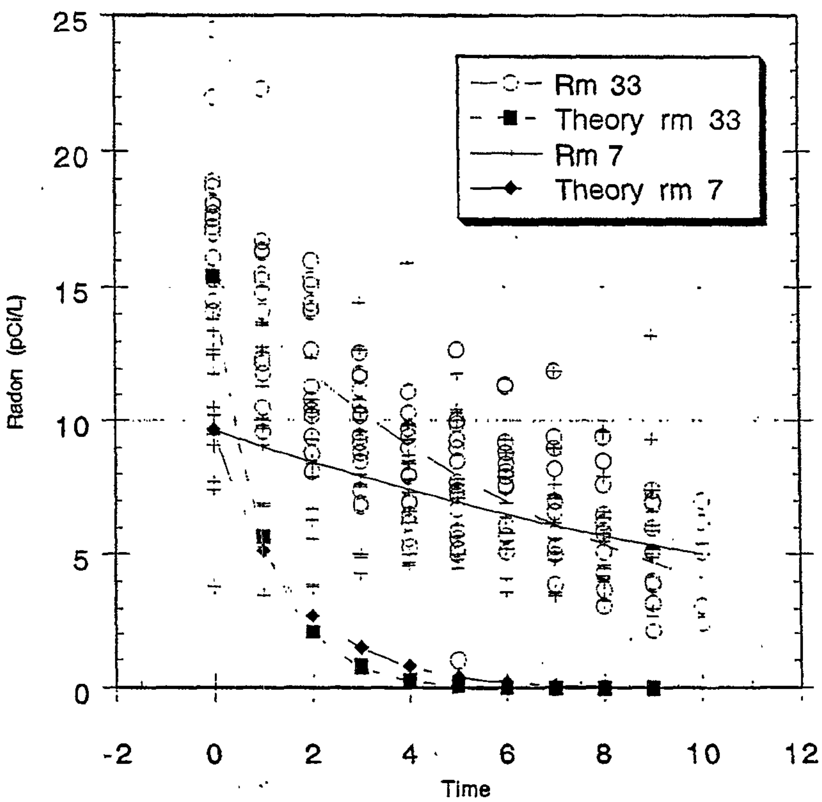
Figure 6 - Reduction Rate of Radon
Air Handlers On - Rms. 7 & 33



y = 15.416 * e^(-0.13157x) R= 0.83454

y = 15.416 * e^(-1x) R= 1

y = 9.6569 * e^(-0.065616x) R= 0.4982

y = 9.67 * e^(-0.63x) R= 1

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are quite different (1 and 0.63 ACH). This is largely due to the difference in source terms. Room 7 begins the average occupied day at around 10 pCi/L while Room 33 begins the average occupied day at just over 15 pCi/L.

It is tempting to attribute the differences in radon dynamics in these two rooms to the difference between exhaust only and fan powered outside air ventilation. But, two rooms, no matter the depth of study provide anecdotal, not conclusive evidence. The results of these measurements do support the current model of radon entry and control as follows :

- entry is dominated by air pressure driven mechanisms
- exhaust ventilation can lower radon concentrations, but not as effectively as powered outside air ventilation

To these two basics we can add a further hypothesis :

- unless fan powered outside air ventilation stops radon entry, the reduction rate of radon will not be as great as expected from dilution alone

and a corollary :

- exhaust only ventilation will never lower radon concentrations as quickly as would be expected from dilution alone because it does not stop the entry of radon

It is important to understand that these two suggestions apply only to dynamic radon behavior and not to steady state conditions. This only applies to the rate at which radon levels change.

<u>Effect Of Outside Air Improvements On Carbon Dioxide Measurements</u>

Introduction--

The reason we breathe is to get oxygen to the cells in our bodies and to remove a number of the byproducts of respiration. Carbon dioxide and water vapor are the most plentiful products of respiration. Carbon dioxide levels in outgoing breath are several thousand parts per million. Carbon dioxide measurements made in occupied rooms can be used as a surrogate for levels of indoor air contaminants that are produced by the occupants themselves and routine activities of occupants. If a simplifying assumption is made about the generation rate of CO_2 being constant then they also can be used to estimate the outside air ventilation rate [4]. The ventilation guidelines of

15 cfm/person in the publication ASHRAE 62-1989 Ventilation for Acceptable Indoor Air Quality should result in a steady state 1000 ppm of carbon dioxide in an occupied classroom.

Carbon Dioxide Measurements--

Carbon dioxide measurements were made in the High School and the Russell School (pre and post radon control) and in the Middle School. The pre radon control measurements were made in early June of 1990 and the post measurements were made in December of 1990.

Carbon Dioxide Measurements in the High School--

A histogram is shown in Figure 7 that differentiates between the pre and post carbon dioxide measurements. Only measurements from occupied rooms with closed windows are shown. The distribution of CO2 levels has been very clearly pushed to the lower levels by the repairs made to the ventilation system. The pre radon control CO2 levels had a mean of 1402 ± 450 ppm and the post level mean was 1042 ± 394 ppm. This represents a 33% decrease in the mean. From a health, comfort and alertness perspective, this is a great improvement over the situation before the ventilation equipment was repaired. Although the mean is now nearly the level recommended in the ASHRAE guidelines[4], half the rooms in the post control sample would still be considered underventilated by the current guideline. Eight percent of them (2 rooms) are above 1700 ppm, which would reflect an outside air exchange rate of 5 cfmiperson. By contrast, all the rooms in the pre mitigation set of measurements were above the current guidelines (1000 ppm) and 27% of them (3 rooms) were above 1700 ppm.

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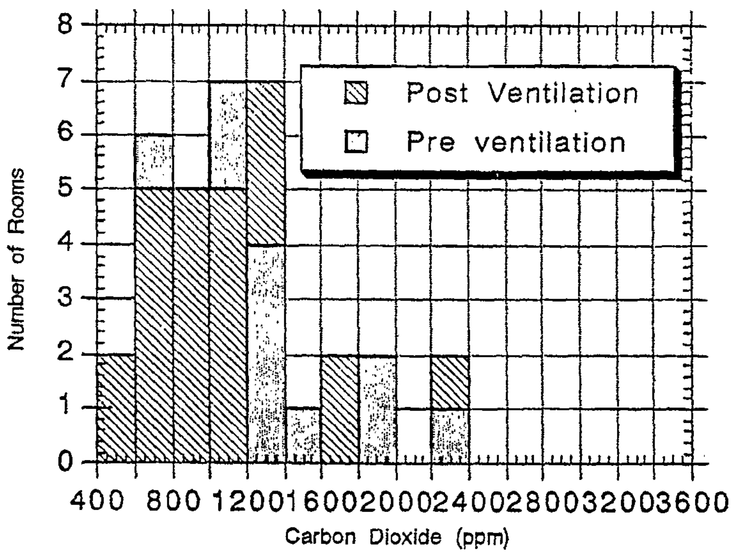
Figure 7 - Pre and Post Control CO2 Histogram for High School



Carbon Dioxide Measurements in the Russell School--

A bar graph is shown in Figure 8 that differentiates between the pre and post carbon dioxide measurements and between ventilation and radon control type. Measurements are from occupied rooms with closed windows except the pre control measurements in the exhaust only ventilation - soil depressurization rooms. These rooms had open windows during the June measurements. The number of open windows is shown on the bar graph.

The CO2 levels have been very clearly lowered by the repairs made to the unit ventilators (rooms 5, 9, and 6) and by the installation of the heat recovery ventilator (located in room 1, with no powered ventilation). Pre control CO2 levels were not available for some rooms with unit ventilators (rooms 7, 8, 10 and 11) but post control measurements were. The mean post control CO2 levels for all the rooms in which unit ventilators were repaired (5, 6, 7, 8, 9, 10, and 11) was 1350 ± 408 ppm.

Rooms 1, 2a, 2b, 3 and 4 are in the oldest wing, where there is no fan powered ventilation. Rooms 2a and 2b show slight increases in CO2 levels, averaging 1500 ppm CO2, as compared to Room 1 which has dropped from over 1250 ppm to 925

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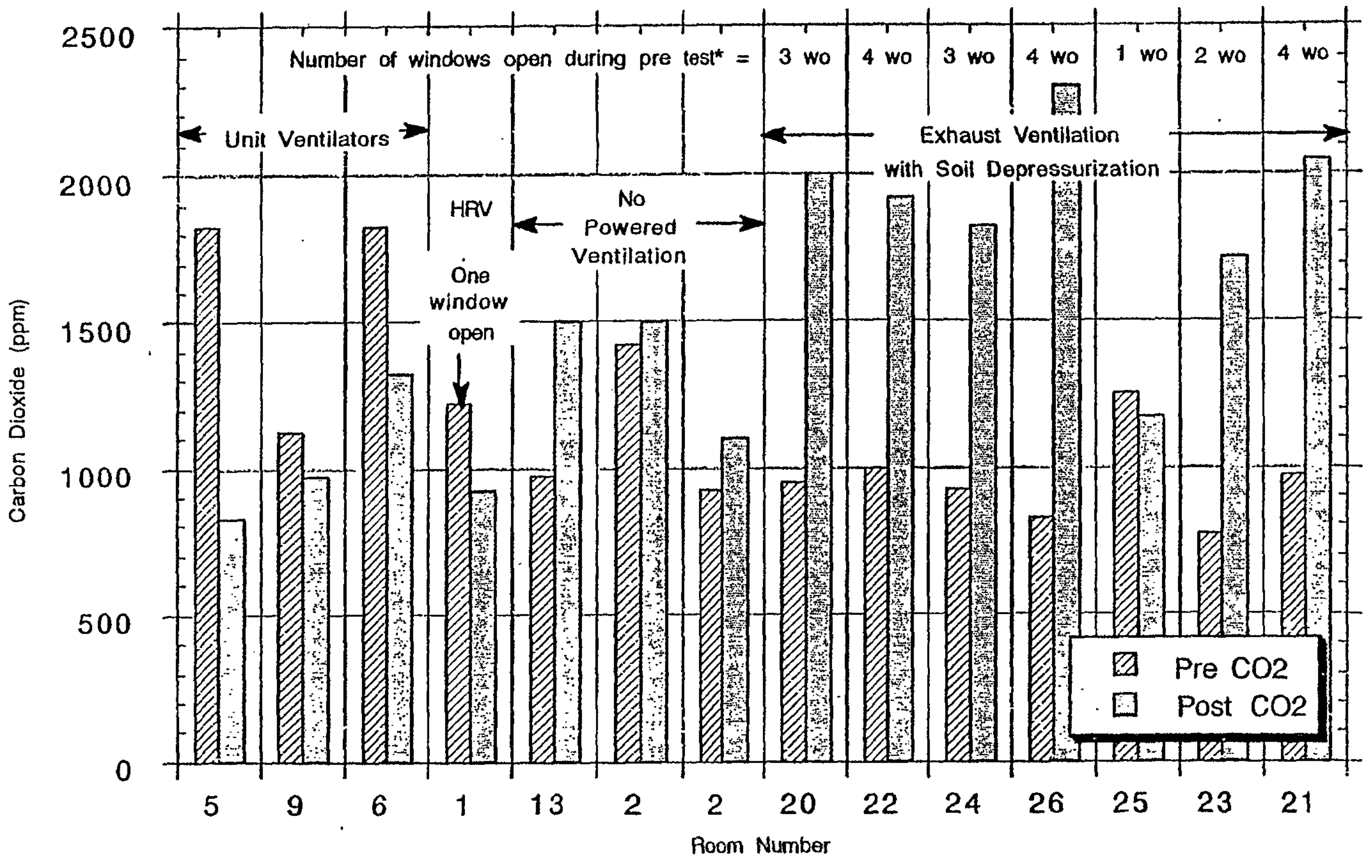

*Note No windows were open in the Unit Ventilator rooms during the CO2 tests

Figure 8 - Carbon Dioxide Levels Pre and Post Radon Control at the Russell School

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ppm. This is expected considering that no changes in the ventilation of rooms 2a and 2b have taken place, but a heat recovery ventilator has been added to Room 1.

Rooms 20, 21, 22, 23, 24, 25, and 26 are in the exhaust only wing, in which soil depressurization has been used to control the radon. The radon levels in these rooms (except for the library, which is around 7 pCi/L) are averaging between 1.4 and 3.5 pCi/L. The pre control CO_2 levels in these rooms must be interpreted cautiously because at least one window was open in each room when these measurements were made. The post control CO_2 levels had a mean of 1857 ± 376 ppm.

None of the exhaust only rooms meet the current ASHRAE guideline for ventilation rates. In fact, none of them meets the ASHRAE ventilation guideline for the year in which they were constructed. While it is clear that soil depressurization will control indoor radon, it is also clear that it has little impact on other indoor air contaminants.

Histograms of the CO_2 data from the Russell School are not presented because there is so little pre control data that did not have windows open.

CONCLUSIONS

Conclusions for this work contribute to interpretation of radon measurements made in school rooms (and other non-residential settings) where a wide range of occupant activities and the operation of air handlers can have important effects on radon measurements. Radon measurements in the Maine Schools show that average radon levels that do not distinguish between occupied and unoccupied conditions can be misleading when the effect of air handlers is unknown.

The operation of both types of air handlers, outside air and exhaust only, has a definite reducing effect on the radon concentrations in the rooms. Unless radon is prevented from entering, the radon concentration does not drop as quickly as expected given the known amount of outside air that is being introduced. Only fan powered outside air has the chance of doing this. In the High School it is not doing so during the coldest months. It is likely that there are times during the spring and fall when the outside air dampers are open wider and the stack effect is reduced that the unit ventilator rooms are pressurized enough to prevent radon entry. Exhaust only ventilation can have reducing effects, but will always be drawing some soil air into the building. It is possible that for given source strengths and slab/building shell leakage characteristics exhaust ventilation could be good enough to control radon, but that is not so in the Gray High School.

Clearly many, if not all the classrooms investigated, were underventilated for the number of occupants. The carbon dioxide data gives plenty of evidence for this contention. Repairing the outside air functions of the air handler made dramatic

improvements in the carbon dioxide levels in the rooms where outside air was introduced. However, while effective and reliable at solving radon problems, soil depressurization in rooms with inadequate ventilation leaves children sitting in high concentrations of CO_2 and other indoor air contaminants for which CO_2 levels are an indicator.

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THE SCHOOL EVALUATION PROGRAM

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ABSTRACT

As part of a coordinated radon in schools technology development effort, EPA's School Evaluation Team has performed on site evaluations of twenty six schools in eight regional locations throughout the United States. This paper presents the results and preliminary conclusions of these evaluations. This represents the largest data bank of schools that have diagnosed with consideration for interactions of the building with both sub slab and heating, ventilating and air conditioning (HVAC) characteristics. Occupied classroom carbon dioxide concentrations and building shell tightness are reported. These measurements help to judge the existing outside air ventilation rates and the potential for building pressurization. Besides these technical issues, physical and institutional problems that affect the selection and implementation of radon control systems in schools are identified. Both soil depressurization and use of existing HVAC equipment were evaluated for each school. Results of this two year study suggest that the EPA should consider a new direction in large building radon abatement - - a holistic approach that considers the broader issue of indoor air quality, comfort, cost and energy issues.

KEY WORDS

Radon, Schools, Large Buildings, Airtightness, Ventilation, Outside Air, EPA, carbon dioxide

INTRODUCTION

The School Evaluation Program (SEP) was originally conceived in the summer of 1989 as a technical assistance program in response to an emerging need for information on diagnostic techniques and mitigation strategies applicable in schools with elevated levels of radon. The program was designed to incorporate all appropriate state of the art radon diagnostic procedures that were successfully being used in residential investigations. Occupant density, and therefore recommended or mandated ventilation rates, is approximately 7 times greater for schools than for residential settings. Accordingly, industry accepted evaluation methods for non-residential air handling systems were incorporated into the program.

The original goal of the SEP was to develop school diagnostic procedures that would consistently provide sufficient information to enable school officials and private sector contractors to choose the most effective mitigation strategy for their school. When completed, the diagnostic procedures will become part of EPA's Technology Transfer Program, and will be made available for training purposes through the Radon Regional Training Centers. Field information collected also would be used to update the EPA school mitigation guidance publication.

The selection of SEP schools was based on four criteria :

1. Schools with radon screening measurements greater than 4 pCi/L.

2. Geographic location/climatic conditions.

3. Structure Type

4. A willingness by the school to mitigate based on the results of the evaluation and the recommended remediation strategy.

As a result of the selection process three schools in Washington state and three schools in New Mexico were evaluated and reported on in a poster paper at the 1990 International Radon Symposium [1]. In fiscal 1990 twenty additional schools were evaluated: three each in the states of Georgia, Iowa, North Dakota, Illinois, and New Jersey, and five in Maine.

The SEP Team in 1990 consisted of the authors of this paper -- a team that represents expertise in the areas of residential radon investigation and large

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building HVAC design, installation, and operation.

The evaluations performed on all twenty six (26) schools have strengthened the investigators opinion and judgement that :

- schools are larger and more complicated in every respect
- all aspects of the building dynamics and purpose must be taken into account when selecting and planning a radon control strategy
- there is a serious problem with low ventilation rates in many of the nations classrooms

An optimum control strategy would depend on each schools' specific needs and would incorporate active sub slab depressurization and HVAC mitigation techniques , independently or collectively, as required.

Through extensive field experience gained by the SEP, a holistic approach to radon control and general indoor air quality in schools become apparent.

METHODOLOGY

The conceptual model for radon entry into buildings has directed field investigations of indoor radon problems toward two areas. The first area is the ways in which the foundation and underlying materials affect radon entry and could be used to control entry by soil depressurization. The second area is an investigation of the ways in which the operation of the mechanical equipment in the building affects radon entry and could potentially be used to control radon entry.

Both approaches, HVAC and soil depressurization control methods prevent soil air entry by managing the air pressure differential relationships between the air in the soil and the air in the building. In addition dilution by increased ventilation plays a role in the HVAC approach and sometimes plays a role in a soil depressurization approach.

RADON ENTRY INVESTIGATION

The radon entry investigation is divided into three parts, review of radon measurements, identification of radon entry points and soil depressurization tests. Each of these are summarized in the following paragraphs.

Radon Measurement Data

Radon measurement data for the schools was obtained through the screening results taken as part of EPA's Phase I and Phase II school survey. Several schools had initiated their own testing program and offered their measurement results for acceptance into the SEP.Most of the schools had tested a many of their classrooms using the current EPA radon measurement protocol. A member of schools had performed confirmation measurements using alpha track detectors.

Radon measurements in schools have been found to vary considerably both spatially and temporally [1]. Figure 1 shows the mean classroom radon levels in the schools evaluated. Error bars of one standard deviation are given to illustrate room to room variation for simultaneous measurements. Temporal variation can be seen by using continuous radon monitors or by the sequential use of passive integrating monitors. These topics will be discussed later in the paper. Both temporal and spatial variation are the result of spatially varying radium concentrations and transport pathways under the building and air pressure differences resulting from the dynamic interaction of occupants, building, mechanical equipment and outside weather conditions [2].

The radon screening measurements for a school were laid out room by room on a floorplan of the school. This can usually be accomplished by using the fire exit floorplans that most public buildings have in hand. The maintenance personnel and the school safety officer proved to be invaluable assets in the investigation of every school. They were a wealth of information about the structure and operation of, and the mechanical equipment within, a school. Often they could provide blueprints for the school and in all cases were able to supply fire exit plans.

By plotting radon screening measurements on the fire exit plans, the pattern of radon levels could be studied. Often this was not particularly enlightening. For example, in many schools, screening measurements ranged between 2 and 8 pCi/L. Given the temporal variation of school room radon levels it is difficult to make a meaningful distinction between a 3 pCi/L screening measurement and a 6 pCi/L screening measurement. This difference might easily occur between measurements made in the same room at different times.

In some schools, the pattern of radon levels was more helpful. For example, a wing in a school that has relatively uniform, elevated concentrations in all the rooms might well have a widely dispersed entry mechanism that the other wings do not. Anecdotal examples are 1) the air handler that supplied conditioned air to the

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classrooms in one zone is mining soil air and delivering with the conditioned air, 2) all wings are built on a permeable sand but two of them have stone pebbles beneath the slab, creating a dilution break at that layer, while one wing has a slab poured directly on the site material providing a radon source at each hole in the slab. Clues like these formed the kick off for the radon source and entry diagnostics, to be covered in the next section.

Radon Entry Points

Many of the schools were slab on grade construction, with a few crawlspaces and basements. The major entry points are summarized in this list :

- joint at the edge of slabs
- water pipe penetrations
- trenches containing heating pipes, water pipes and drains
- trenches used as conditioned air supply and returns
- crawlspaces with all of the above

The radon concentrations beneath the slabs of the schools were measured. The results are shown in Figure 2, with a summary of the HVAC equipment in the schools. As can be seen they varied from a low of 200 pCi/L to a high of 8000 pC/L with a mean of 1500 pCi/L. While high compared to all but the most extreme cases of indoor levels these are relatively low sub slab concentrations for many residential buildings with elevated radon.

Soil Depressurization Tests

In order to assess the potential for radon control by soil depressurization, a vacuum suction test was made in most schools. This test allowed visual identification of the sub slab material and a measurement of how easily air could be drawn from beneath the slab. Figure 2 has a column that lists the type of sub slab material and another that lists the amount of air that could be pulled from under the slab using the vacuum. Generally speaking, the lower the amount of air that can be drawn from under the slab, the more difficult it is to extend a low pressure field beneath the slab [3]. Low airflows, consequently would mean more suction points for a successful soil depressurization system. In a few schools the vacuum could not compete with the suction put on the building by the exhaust fans.

HVAC CHARACTERIZATION

System Operation

Review of system plans is the first step in evaluating the expected effect of the HVAC system's effect on radon. Equipment observations must be performed in order to determine just what equipment is actually being operated, its operation schedule, the control sequence, and wether the air flows are near the design quantities. Air flow observations may be as simple as visually observing the position of an outside air damper blade or the operation of an exhaust fan and the direction of air flow with a chemical smoke pencil, or may be more involved. Measurement of actual exhaust flow rates with an air balancing hood has proven valuable for determining the operational status of exhaust systems and for conducting a building shell tightness test. Observations of closed outside air intakes or inoperative make-up air supply fans are typical indicators of HVAC system not being operated or maintained as they were design to be [3].

The HVAC systems in the SEP schools were characterized in terms of heating and cooling, ventilation, control strategy and the potential impact on radon levels. A wide variety of ventilation systems were found in the school buildings. The HVAC features are summarized in Figure 2.

A summary of the HVAC characteristics is as follows:

- 96% mechanical ventilation system
- 38% single ventilation type
- 27% three or more ventilation types
- 12% no mechanical outdoor air supply

One of the prominent features of the HVAC systems was the number of problems found. These problems covered a range that included inoperative equipment (broken belts, fans, controls), equipment that had never been wired, poorly maintained equipment (disabled damper linkage, dampers painted shut), poorly designed equipment (ventilators too small or not used because they are too noisy and the control is by teachers) and unwitting modification of the ventilation system (replacing the rolled steel sash window walls with insulated wall). The extent to which these types of problems were present was remarkable. **Every** school visited suffered from at least one of these problems.

Ventilation Air Delivery Rate

A continuous carbon dioxide monitor was used as an indicator of ventilation rates. In an occupied classroom the CO_2 level is a function of the number of students and the ventilation rate. These measurements are invaluable when deciding whether a radon control approach that increases the ventilation rate is appropriate or not. Carbon dioxide measurements were made in the classrooms in the mid afternoon before students were let out. Levels over 1000 ppm indicate that the current ASHRAE guideline [4] of 15 cfm (7l/s) per student is not being met. Figure 3 shows CO_2 levels made in occupied classrooms of nine schools. Most rooms in these schools were above the ASHRAE guideline. The mean CO_2 level was 1780 ppm for this sample of rooms. Figure 4 shows radon screening measurements plotted versus the CO_2 levels for a number of rooms. It can be seen from this data that a room that has elevated CO_2 levels may not have elevated radon levels and vice versa. This chart should be veiwed with caution as the radon measurements were made at a different time and in different mode than the carbon dioxide measurements. This data forms the basis for the SEP Team feeling that broader indoor air quality issues need to be considered when investigating radon in schools.

Building Shell Pressure Relationship

A measurement of the pressure relationship between the inside of the building and outside is one of the key parameters which needs to be investigated. This can be accomplished on a not too windy day by simply making measurements through a crack in a closed doorway or window utilizing a sensitive electronic micromanometer (pressure transducer). Readings of slight positive pressure (+.001 to +.010 inches water) will help to keep radon out during operation of the HVAC system, while negative readings will cause increased radon entry.

Building Shell Tightness Test

To determine how much make-up air would be needed to slightly pressurize a building, the equivalent of a fan door pressure test can be performed. The results of this test will reveal the practicality of "building pressurization" to mitigate an observed radon problem. In the schools this test was performed on they all fit within the normal leakage areas for buildings of this size.

RECOMMENDATIONS

In each of the school buildings both a soil depressurization and an HVAC approach to controlling indoor radon levels was addressed. An HVAC system

approach was considered the first choice in 23 of 26 schools. This high fraction reflects the number of schools whose ventilation rates did not meet current guidelines and whose radon levels were low enough that meeting them likely control the radon. Soil depressurization was often listed as a second option in the event that increasing the ventilation rate to meet guidelines did not lower the radon sufficiently or if the school needed to respond to the radon levels more quickly than modifying the HVAC system would allow.

Note : It is important that any approach that is used in school buildings comply with state requirements for qualifications in the design and installation of mechanical systems. Any HVAC work should involve a professional engineer experienced in air handling systems and their interaction with the building shell.

CONCLUSIONS

Conclusions form this work are :

- school rooms do have elevated indoor levels
- there appears to be a serious problem with under ventilated schoolrooms in US schools
- radon in school rooms must be seen in light of other health concerns
- in order to use HVAC systems to control radon in schools a team approach must be used that incorporates school maintenance, professional engineers and a radon professional. These are not necessarily different people.
- interpreting radon measurements made in classrooms is difficult because of the wide variety of activities that occur in schools.

It is the opinion of the authors that the current underventilated status of many school rooms is the result of an unwillingness of Americans to fund school design, construction and maintenance at a level that would ensure adequate ventilation for the children. There is a litany of specific technical problems, but the underlying theme is lack of knowledge and funds. If our nation knew that our children were trying to learn in situations where the air they breathe is contributing to drowsiness, poor focus of attention and headaches I believe this would be remedied. Americans, for all our faults are generous people.

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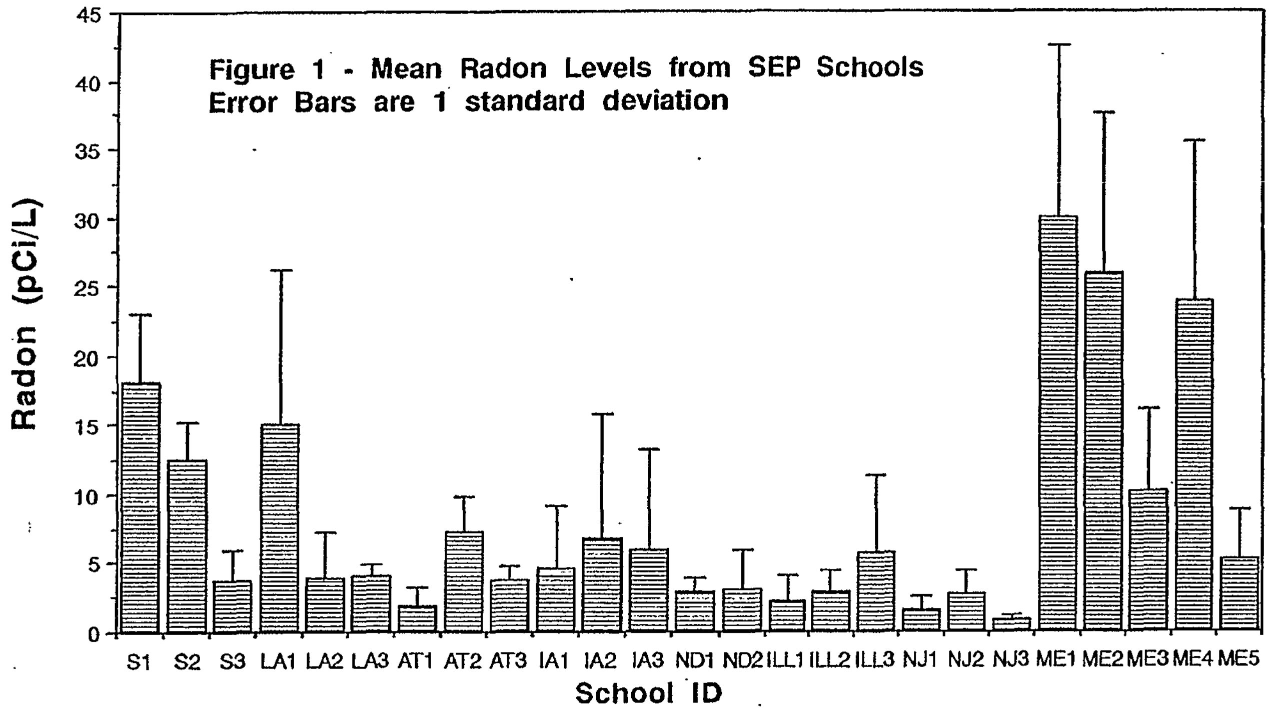

Figure 1 - Mean Radon Levels from SEP Schools
Error Bars are 1 standard deviation

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Figure 2 - Summary of School HVAC and Sub Slab Characteristics

			Ventilation (cfm) (measured)				Sub Slab		Vac.	Window	Floor	Num.	Num.
Sch. ID	Heating	Cooling	Exhaust	Make up	cfm/per.	Control	Material**	Rn pCi/L	cfm	Retrofit?	Area	Student	Staff
S1	Warm air	AC	1000*	170/OA	10	Tstat	FS	600±200	6				
S2	Hydronic	None	5800*	OA/off	11	Tstat	CS	1100±200	27				
S3	Warm Air	AC	NT	VAV	NA	CPU man.	NT	NT					
LA1	Hydronic	None	10000	400/UV	14	Tstat	S&G	500±50	20				
LA2	Hydronic	None	NT*	None	NA	Tstat**	SP	900±100	38				
LA3	Warm Air	AC	NT*	NT/UV	NA	Tstat'	NT	NT					
GA1	HP WA	HP	3600*	OAF/off	5	Tstat/Clock	SP(1/4")	800	43	unk.	49390	680	40
GA2	HP WA	HP	NT/inop.	OAF/off	NA	Tstat/Clock	SP/Clay	1220-8000	25	unk.	44251	unk.	unk.
GA3	Warm Air	AC	NT/inop.	OA/off	NA	Tstat/Clock	SP	4000±1000	40	unk.	60816	729	61
IA1	Hydronic	None	2000	Wind.	5	Man.	FS/Clay	700±200	2~16	blocked	26000	215	25
IA2	Hydronic	Wall AC	400*	Wind.	3	Man.	FS	500±200	18	unk.	14000	110	10
IA3	Hydronic	Wall AC	5000	Wind.	34	Man.	S&G	1300±200	17~40	retrofit	67564	115	30
ND1	Warm Air	None	2300	OA/off	6	Tstat/Man.	CS	800±500	20	blocked	47500	350	36
ND2	Steam	None	8800	UV/Wind	18	Tstat/Man.	SP	600±100	17~46	retrofit	60800	400	27
ND3	Many	None	NT	OA/HRV	NA	Tstat/Clock	gap	700±150	46	unk.	largest	unk.	unk.
ILL1	Warm Air	None	6357	OA/UVoff	14	Tstat/Clock	gap	200±100	60	blocked	41570	430	20
ILL2	Steam	None	5100*	OA/UVoff	10	Tstat/Clock	FS	700±150	7	no	36000	507	19
ILL3	Hydronic	None	NT*	OA/UVoff	NA	Tstat/Clock	Crawl	20	NA	limited	28000	328	13
NJ1	Hydronic	AC	NT	OA/UVoff	NA	Tstat/Clock	S&G/SP	2000/360	46	no	58900	457	68
NJ2	Hydronic	AC	NT	OA/UVoff	NA	Tstat/Clock	CS,SP	1500/300	20	no	27752	400	30
NJ3	Hydronic	AC	NT	OA/UVoff	NA	Tstat/Clock	SP	150±20	NA	no	32500	480	50
ME1	Hydronic	None	5800	UVoff	13	Tstat/Clock	S&G	4500±1500	2&42	no	34000	374	44
ME2	Hydronic	None	Bill	OA/UVoff	NA	Tstat/Clock	S&G	3500±1000	46	no	106500	540	46
ME3	Electric	None	11300	OA/UVoff	9	Tstat/Clock	S&G,SP	3500±2000	10~45	no	167000	1200	60
ME4	Hydronic	None	NT	UVoff	NA	Tstat/Clock	SP	2500±1000	32	no	10500	85	
ME5	Steam	None	NT	OA/UVoff	NA	Tstat/Clock	S&G	2500±1000	25	no	120000	unk.	unk.

* many fans inoperative Note : The Kitchen Exhaust is included in the total for most schools making cfm/person high

** an installed enrgy management control had been disabled

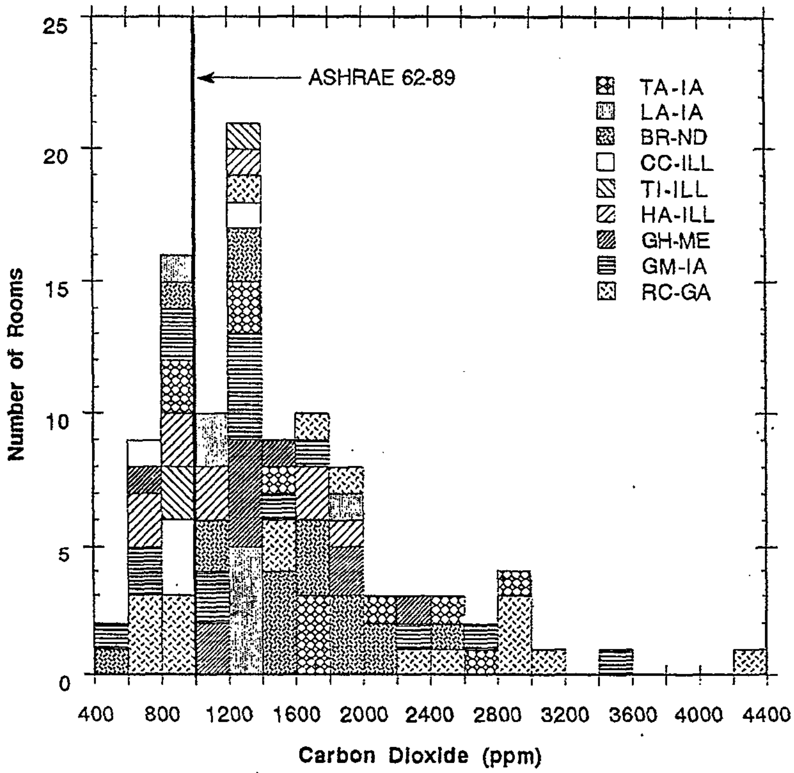
' the controls for the Unit Ventilators were in each classroom and teachers reported they didn't operate them because they were too noisy

** FS = Fine Sand CS = Coarse Sand S&G = Sand and Gravel

SP = Stone Pebbles NT = Not Tested

B.30

Figure 3 - Histogram of Carbon Dioxide Measurements Made in Occupied Classrooms SEP-1990



Note : Measurements made in rooms with open windows or less than five people are not included in this data set.

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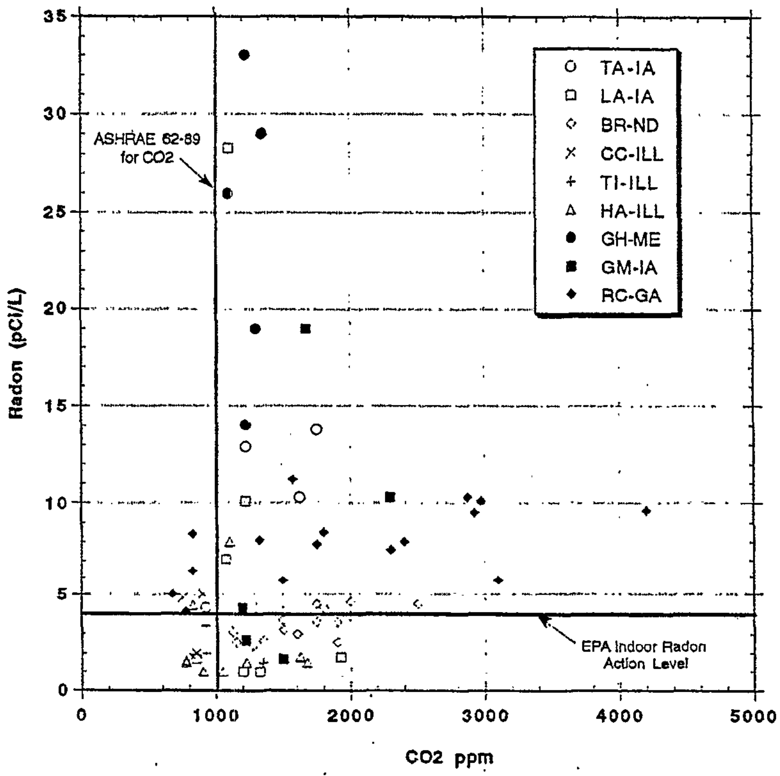
Figure 4 - Room Carbon Dioxide vs. Radon Screening Measurements in School Rooms



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THE UNIVERSITY OF TULSA
Division of Continuing Education
The Center for Environmental Research and Technology

The United States Federal EPA IAQ Research Program

Speaker:

KEVIN TEICHMAN, Ph.D.

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Office of Technology Transfer & Regulatory Support (OTTRS)
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The U.S. Federal Indoor Air Quality RESEARCH Program

Kevin Teichman, Ph.D.
Office of Research and Development
U.S. Environmental Protection Agency

Office of Technology Transfer & Regulatory Support (OTTRS)
Office of Research & Development (ORD)

COMMITTEE ON INDOOR AIR QUALITY (CIAQ)

Cochair Agencies

Environmental Protection Agency
Department of Health and Human Services
Department of Energy
Consumer Product Safety Commission

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Office of Technology Transfer & Regulatory Support (OTTRS)
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ENVIRONMENTAL PROTECTION AGENCY (EPA)

Indoor Air Quality Research Groups

- o Engineering
- o Monitoring
- o Health Effects
- o Exposure Assessment and Risk Characterization

Office of Technology Transfer & Regulatory Support (OTTRS)
Office of Research & Development (ORD)

ENVIRONMENTAL PROTECTION AGENCY (EPA)

Indoor Air Quality Research Groups

- o Air and Energy Engineering Research Laboratory (AEERL)
- o Atmospheric Research and Exposure Assessment Laboratory (AREAL)
- o Health Effects Research Laboratory (HERL)
- o Environmental Criteria and Assessment Office (ECAO/RTP)

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Office of Technology Transfer & Regulatory Support (OTTRS)
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EPA ENGINEERING RESEARCH

Air and Energy Engineering Research Laboratory (AEERL)

- o Chamber Studies of Organic Emissions from Indoor Material Sources
- o Test House Studies of Material, Combustion, and Activity Sources
- o Indoor Pollutant Control Techniques
- o Radon Mitigation Research

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EPA MONITORING RESEARCH

Atmospheric Research and Exposure Assessment Laboratory (AREAL)

- o Total Exposure Assessment Methodology (TEAM) Field Studies
- o Monitoring and Methods Development
- o Indoor Air Quality Modelling
- o "Sick Building" Field Studies

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EPA HEALTH EFFECTS RESEARCH

Health Effects Research Laboratory (HERL)

- o Neurobehavioral and Sensory Irritant Effects of VOC Mixtures
- o Evaluation of Cotinine as a Biomarker of Environmental Tobacco Smoke Exposure
- o Effects of Indoor Pollutants (NO2 and ETS) on Pre-School Children

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EPA HEALTH IMPACT RISK ASSESSMENT RESEARCH

Environmental Criteria and Assessment Office (ECAO/RTP)

- o Evaluation of Different Approaches to Risk Characterization
  - Pollutant Mixtures
  - Non-Cancer Health Endpoints
- o Program Management and Technology Transfer
  - Indoor Air Information Assessment
  - Indoor Air Reference Data Base

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Office of Technology Transfer & Regulatory Support (OTTRS)
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DEPARTMENT OF HEALTH AND HUMAN SERVICES (DHHS)

National Institute for Occupational Safety and Health (NIOSH)

- o Health Hazard Evaluations
- * o IAQIV Performance in Large Buildings (Epidemiologic studies)

Office of Technology Transfer & Regulatory Support (OTTRS)
Office of Research & Development (ORD)

DEPARTMENT OF HEALTH AND HUMAN SERVICES (DHHS)

- o Measurement of Biochemical Indicators of Tobacco Exposure (CDC)
- o Role of Dust Mite, Cockroach, and Cat Allergens in Houses/Asthma (NIH)
- o Radon Epidemiologic Studies (NCI)

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Office of Technology Transfer & Regulatory Support (OTTRS)
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DEPARTMENT OF ENERGY (DOE)

- o Indoor Radon 10 million/yr
- o IAQ Control Techniques
- o Infiltration, Ventilation, and IAQ in Large Buildings
- o Energy Performance of Buildings

Office of Technology Transfer & Regulatory Support (OTTRS)
Office of Research & Development (ORD)

CONSUMER PRODUCT SAFETY COMMISSION (CPSC)

- o Combustion Emissions from Kerosene and Unvented Gas Space Heaters
- o Chamber and Field Studies of Biological Aerosols
- o VOC Emissions from Consumer Products, e.g., Methylene Chloride

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OTHER AGENCIES PERFORMING IAQ RESEARCH

- General Services Administration (GSA) - three new fed bldg. epi studies will be done
- Housing and Urban Development (HUD)
- National Institute of Standards and Technology (NIST)
- Bonneville Power Administration (BPA) utility

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FUTURE DIRECTIONS

- In August, 1989, EPA submitted its "Report to Congress on Indoor Air Quality," which stated that "sufficient evidence exists to conclude that indoor air pollution represents a major portion of the public's exposure to air pollution."
- The Report to Congress also presented six recommendations that identify future activities for both the public and private sectors.

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IAQ REPORT TO CONGRESS RECOMMENDATIONS

1. Expand research devoted to characterizing exposure and health effects of chemical contaminants and pollutant mixtures commonly found indoors.
2. Develop a research program to characterize and develop mitigation strategies for biological contaminants.

Office of Technology Transfer & Regulatory Support (OTTRS)
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IAQ REPORT TO CONGRESS RECOMMENDATIONS

3. Expand research devoted to identifying and characterizing significant indoor air pollution sources and evaluating appropriate mitigation strategies.
4. Develop and promote, in conjunction with appropriate private sector organizations, guidelines for ventilation, as well as other building design, operation, and maintenance practices to ensure that indoor air quality is protective of public health.

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IAQ REPORT TO CONGRESS RECOMMENDATIONS

5. Expand current efforts to provide technical assistance and information dissemination to State and local governments, the private sector, and the public.

6. Undertake to characterize the nature and pervasiveness of the health impacts associated with indoor air quality problems in different building types, and promote guidelines for diagnosing and controlling such problems.

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IAQ RESEARCH NEEDS

- Risk Assessment
- Exposure Assessment and Modelling
- Source-Specific Research
- Building Systems
- IAQ Control Techniques
- Crosscutting Research
- Technology Transfer

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IAQ RESEARCH NEEDS

Risk Assessment

- o Develop risk methodology procedures
- o Perform assessments for major indoor air pollution scenarios.

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IAQ RESEARCH NEEDS

Exposure Assessment and Modelling

- o Develop screening protocols, questionnaires, and measurement methods for complaint-building studies
- o Evaluate and validate new measurement methodologies, under field conditions, for aerosols, organics, biological species, and air exchange rates
- o Further develop spatial/temporal models, source models, receptor models, and exposure models for indoor environments

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IAQ RESEARCH NEEDS

Source-Specific Research

- Characterize emissions from unvented combustion appliances
- Measure emission rates of organic compounds from building materials, furnishings, and consumer products
- Perform health effects research devoted to both cancer and non-cancer health risks from combustion sources

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IAQ RESEARCH NEEDS

Radon

- Continue mitigation research for new and existing houses, and initiate research devoted to other building types, e.g., schools
- Initiate the testing of novel materials and devices to reduce radon progeny exposures
- Perform geologic/geographic assessments of regions potentially at high (or low) radon risk
- Conduct fundamental health effects research, including animal inhalation and epidemiologic studies

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IAQ RESEARCH NEEDS

Biological Contaminants

- Initiate development of standardized monitoring methods
- Identify and establish baseline concentrations of major classes of biological contaminants
- Investigate the contribution of HVAC equipment to indoor levels of biologicals

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IAQ RESEARCH NEEDS

Control Techniques

- Test and evaluate the effectiveness of source modifications, including changes in product composition and use, conditioning of building materials before use, and product substitution
- Conduct both laboratory and field studies to determine the effectiveness of air cleaners

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IAQ RESEARCH NEEDS

Building Systems

- o Develop techniques and protocols to measure ventilation effectiveness
- o Measure ventilation rates and ventilation effectiveness in complaint-building investigations and residences
- o Conduct an integrated assessment of the combined impacts of source emissions, pollutant levels, ventilation rates, and energy consumption in new building designs, and perform follow-up measurements

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IAQ RESEARCH NEEDS

Crosscutting Research

- o Conduct studies regarding the prevalence of building-occupant symptoms and indoor pollutant levels
- o Conduct an epidemiologic study of the impact of indoor air quality on productivity
- o Conduct ergonomic and psychosocial research

– Tschman's dream!

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IAQ RESEARCH NEEDS

Technology Transfer

- Promote technical information transfer among Federal agencies, states, and the private sector, e.g., compendium of indoor air test methods
- Work with the program office to publish public information materials, e.g., the "Inside Story: A Guide to Indoor Air Quality"
- Cosponsor IAQ conferences and workshops

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Biography of Dr. Kevin Teichman

Dr. Kevin Teichman is a Supervisory Environmental Engineer in the U.S. Environmental Protection Agency's Office of Research and Development (ORD). His responsibilities in this position include serving as the Chief of the Air Team within the Office of Technology Transfer and Regulatory Support. In this capacity, Dr. Teichman is responsible for coordinating ORD participation in EPA's policymaking activities related to air and radiation pollution, and helping to develop ORD's research program in these areas. In addition, Dr. Teichman is the Headquarters coordinator of EPA's indoor air quality research program. For example, during 1989, he participated in the preparation of EPA's "Report to Congress on Indoor Air Quality."

Dr. Teichman's prior work experience includes serving as the program manager for both the Department of Energy's indoor air quality, infiltration, and ventilation and performance calculations research programs; as a Senior Mechanical Engineer for the architectural/ engineering firm Ellerbe Associates; and as an Assistant Professor of Mechanical Engineering at the University of Minnesota. He holds B.S. and M.S. degrees from M.I.T. and a Ph.D. degree from the University of California at Berkeley -- all in mechanical engineering.

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THE UNIVERSITY OF TULSA
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The Center for Environmental Research and Technology

Residential IAQ Problems

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THAD GODISH, Ph.D.

Dr. Thad Godish is Director of the Indoor Air Quality Program at Ball State University's Office of Research & Sponsored Programs, Indianapolis, Indiana. He is also the author of the critically acclaimed book ***Indoor Air Pollution on Control***, 1989, and he works as a consultant for Indoor Air Quality Services.

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pulmonary Effects
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Cancer
asphyxiation - low level CO poisoning

Combustion Sources - No 1 problem
* flue gas spillage - CO - not pure CO
upper resp symptoms

Formaldehyde Sources -
95% comes from wood products - with urea bonded
hardwood plywood paneling urea formaldehyde
particle board resin
pressed wood

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Current Legal Trends on Indoor Air

Speaker:

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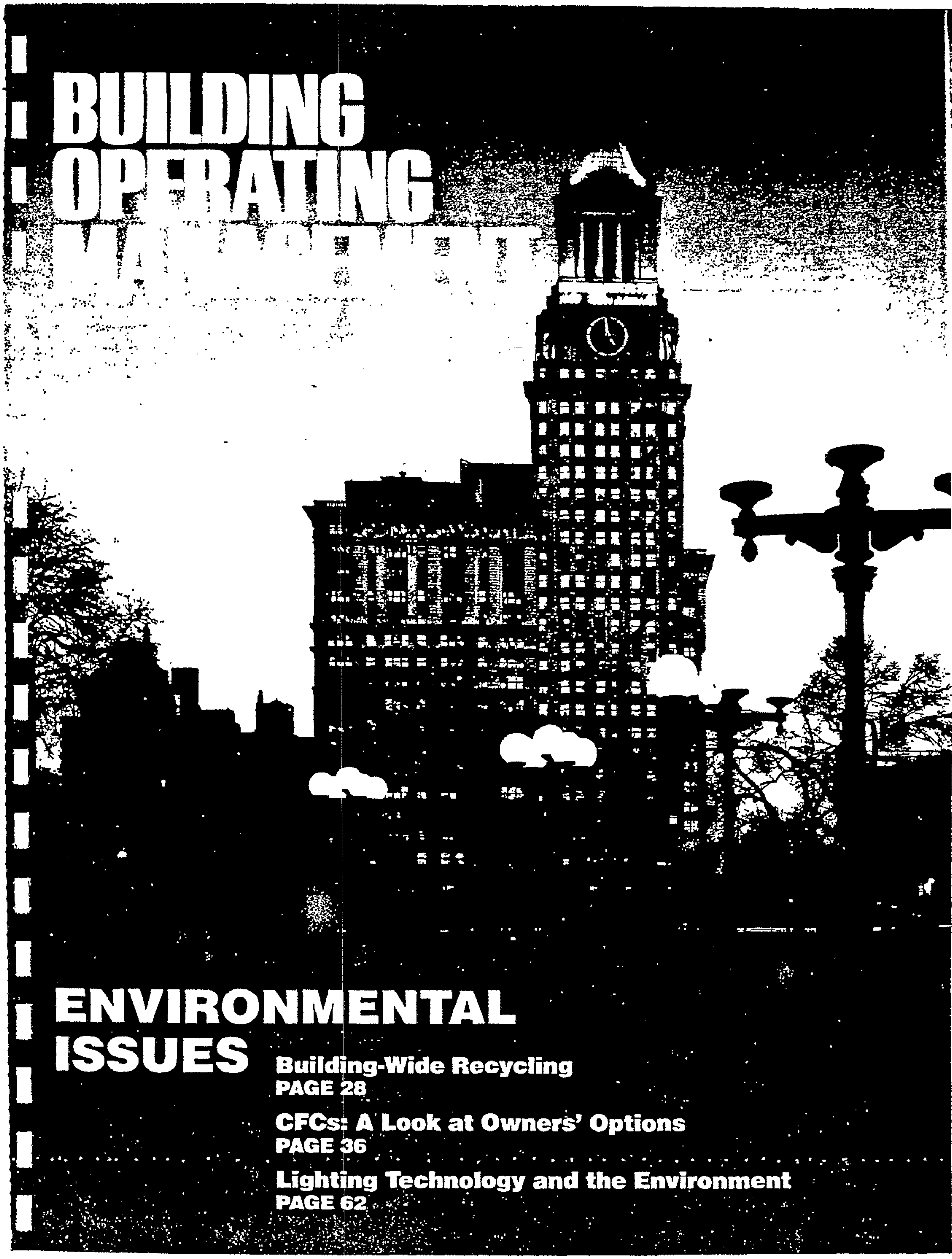
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PROTECTING OCCUPANTS – AND OWNERS – FROM INDOOR AIR PROBLEMS

As lawsuits related to sick building syndrome rise, owners find that improving air quality reduces liability

by LAURENCE S. KIRSCH and BONNIE Y. HOCHMAN

As public attention has focused on indoor air quality, building owners have learned of reported claims of health problems experienced by building occupants. What some building owners may not know, however, is that allegations of indoor air quality problems can be unhealthy for themselves as well — even if they never set foot in the building.

Complaints concerning the quality of the indoor air can have a significant effect on the financial health of even the most prosperous owners. This is the case because building owners face an increasing risk that they will be the target of a lawsuit by a building occupant charging that myriad health effects — potentially ranging from headaches to allergic sensitization to miscarriages — have been caused by poor indoor air quality.

During the past several years, building occupants have filed an increasing number of lawsuits of just this kind, claiming they are suffering from "Sick Building Syndrome" (SBS). And unable to establish that any particular party is at fault, sick building plaintiffs and their attorneys have chosen to sue a large number of parties — including building owners, architects, contractors, manufacturers of products used in buildings, and even the companies that have tested and attempted to mitigate indoor air quality problems.

In a typical sick building case, individuals in the same building believe they are experiencing similar symptoms — such as coughing, headaches, respiratory irritation, dizziness or nausea upon entering the building — and that such symptoms are associated with their presence in the building.

Such symptoms may be caused by improper ventilation, bacteria or other microorganisms in a ventilation system, ventilation of outdoor pollutants into the building through air intake ducts, chemicals used in the office environment or even the materials comprising the building itself or its furnishings. These symptoms, however, could also be caused by generalized worker dissatisfaction or the flu.

Limited worker's compensation

Thus, sick building cases are difficult to prosecute and just as difficult to defend. Sick building lawsuits are being directed at building owners because state workers' compensation laws typically bar workers from suing their employers directly for work-related health conditions. The money a worker can recover under a worker's compensation program, even if recovery is allowed, are limited.

Thus, employees who believe that they have been injured by sick buildings have felt compelled to seek alternative means of recovery against parties other than their employer. Unfortunately, building owners can be convenient targets.

From the perspective of the building owner such lawsuits can be horribly unfair. Few owners would intentionally allow their buildings to be maintained in an unsafe manner, and in most buildings that have been found to contribute to occupants' health problems, the owners have been operating these buildings in full compliance with federal, state and local law. Under these circumstances, it is questionable why owners should be forced to defend themselves against sick building lawsuits.

The potential bases for these lawsuits vary almost as much as the potential defendants. Indoor air pollution cases have relied on theories such as negligence, breach of express or implied warranties, strict liability, landowner/occupier liability, assault, battery, nuisance, infliction of emotional distress, misrepresentation, fraud, as well as claims for other equitable relief.

The following sections will first discuss certain examples of indoor air litigation, of which building owners should be aware, and next provide certain suggestions for avoiding or minimizing the damage caused by sick building lawsuits.

SBS lawsuits

One recent Iowa case illustrates many of the difficulties inherent in sick building cases. *Bloomquist v.*

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Wapello County, et al. is the first sick building case to ever reach a jury. In *Bloomquist*, a jury awarded $1 million dollars to three workers and their spouses to compensate them for injuries allegedly suffered as a result of repeated exposure to various pesticides in an office with a faulty heating, ventilation and air conditioning (HVAC) system. After the trial, however, the judge rejected the jury's verdict and issued a directed verdict in favor of the defendants on the ground that the plaintiffs had failed to prove that either the pesticides or the faulty HVAC system had caused the injuries sustained.

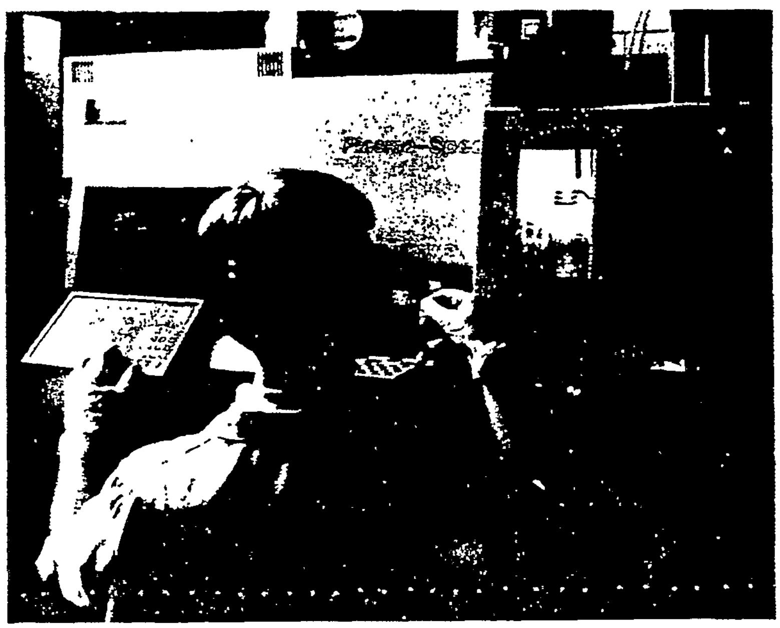
In another case, which has withstood motions for summary judgment, a Los Angeles trial court this fall will examine allegations of negligence, strict liability, implied and express warranties of fitness and merchantability, fraud, conspiracy and breach of covenant of quiet enjoyment.

In *Call v. Prudential Insurance Co.* several tenant corporations and their employees sued the landlord, building owner, architects, engineers, contractors and 250 unknown defendants to recover personal as well as business injuries that they said resulted from SBS. The plaintiffs claimed that the HVAC system was faulty and the building was permeated by toxic fumes, chemicals and substances.

One of the first lawsuits filed as a result of SBS was *Buckley v. Kruger-Benson-Ziemer*. In this case, a computer programmer sued approximately nine named and 280 unnamed defendants for the injuries he allegedly sustained from exposure to indoor pollutants in his poorly ventilated, tightly enclosed workplace.

The plaintiff said that the various defendants — including architects, contractors, mechanical engineers, heating and air conditioning consulting engineers, manufacturers, distributors, sellers and installers of the air conditioning equipment, carpentry and floor tiles as well as the manufacturers, sellers and distributors of "certain chemicals commonly used in offices, including but not limited to toners used in duplicating machines" — knew or reasonably should have known and had a duty to warn all persons including the plaintiff of the poor air ventilation and the dangerous chemicals and ▶

Owners may feel the effects of steps taken — or not taken — to protect building occupants from poor indoor air quality. Unclean air ducts might be evidence of a lack of concern, while testing might show an attempt to prevent health problems.



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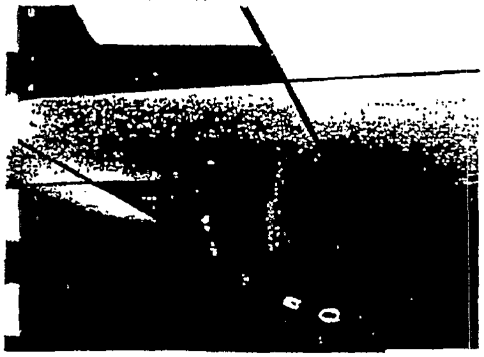

Regular duct cleaning can help prevent air quality problems.

INDOOR AIR QUALITY

toxins in the air, carpet, tile and office machinery.

The plaintiff claimed liability based upon negligence, strict liability and infliction of severe emotional distress. The case finally settled before trial for a purported $622,500, but only after extensive, protracted discovery.

Similarly, in *Stillman v. South Florida Savings & Loan*, a bank tenant asserted indoor air pollution-related counterclaims against a landlord after vacating an allegedly sick building and being sued for breach of lease. The bank asserted that it vacated the building after having demanded that the landlord investigate and alleviate the indoor air pollution that was causing its employees to suffer from SBS.

The bank claimed that the landlord's failure to maintain properly the air conditioning system was a breach of the bank's lease. The bank also asserted that the indoor air quality problems had breached the bank's right to the quiet enjoyment of its premises, constituted a constructive eviction from the property, and was the result of the landlord's negligence. Three years after being filed, this case remains unresolved.

In *Henley v. Blomfeld Co.*, several state employees sued the architect, contractors and owner of an office building to recover damages for the personal injuries they suffered from the alleged microbiological contaminants in the carpeting and HVAC system.

While the building had been investigated for indoor air pollution by the State Department of Labor in 1985 after a more than five-year occupancy, and 11 employees were removed due to the severity of their symptoms, the building was not evacuated until February of 1986 when the problem allegedly became so severe that an employee collapsed at her desk.

The allegations are based on strict liability, negligence, recklessness and breach of express and implied warranties. This case was recently settled on undisclosed terms.

These are only a few examples of the numerous lawsuits that have been brought under sick building-type theories, and illustrate both the broad scope of potential liability and the pressures to settle that can be inherent in such cases.

How building owners can protect themselves

Given the significant potential for liability that may be encountered from sick building claims, it would seem prudent for building owners to take measures to protect themselves. Through the following general suggestions, which must be evaluated carefully with counsel in the context of each specific situation, building owners may help avoid finding themselves as the target of a sick building lawsuit or, if they are sued, may help limit their potential liability:

1) Inform responsible employees about SBS and establish a focal point to which all indoor air complaints should be directed. Building owners cannot protect themselves against a sick building complaint if they do not learn of it in a timely manner. Many employee, customer or tenant complaints are not taken seriously because they are directed to individuals who lack knowledge about the problem or lack authority to cure it. Moreover, if complaints by different building occupants are directed to different management personnel, the building owner may not learn of any emerging patterns.

Implementing a process which ensures that SBS complaints are directed to specified individuals who have knowledge and authority to respond may alleviate the escalation of problems.

2) Take pre-emptive measures where possible. The best way to limit liability is to discover and remedy a potential problem before complaints arise. Indoor air testing and diagnosis not only eliminate problems before they develop, but also may serve as evidence of reasonable efforts to guard against poor indoor air quality.

3) If any problems are discovered, take steps to deal with and document them. Just as pre-emptive measures may be used as evidence of non-negligence, an owner's failure to address a known existing air pollution problem may be used as evidence to support a punitive damage claim. If a problem becomes apparent, it is important that an owner take steps to remedy it. A record of the mea- ▶

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sures taken should be kept.

4) Indoor air quality issues should be considered during the performance of regular building management activities. Certain activities — construction, renovation, operating ventilation systems or manufacturing products — may affect indoor air quality. By considering the potential impact of these activities, an owner can most likely avoid problems.

5) Provide for contractual protection. Where it is appropriate, seek contractual protection, such as releases and indemnities from tenants, suppliers or subcontractors.

6) Keep informed of standards and guidelines and comply with them. Compliance with standards may provide a shield from liability. By participating in the efforts of trade associations and organizations in developing voluntary standards, building owners will be better informed of the rapidly developing field of indoor air quality. In addition, they will be better able to document their reasonable care in addressing indoor air quality issues.

7) Recognize legal problems and seek legal advice. Given the scientific, medical and technical aspects of indoor air quality issues, it is easy to overlook their legal aspects. Indoor air complaints have the potential to escalate into serious legal problems, and such complaints should be treated accordingly. By seeking competent legal advice when faced with an indoor air complaint, an owner may immediately begin taking appropriate steps to protect his or her interests..

For example, a lawyer may be able to assist in the proper handling of the complaint and in the handling of similar complaints from other employees. A lawyer can help protect a building owner from making potentially damaging admissions based on preliminary information that may prove incorrect. The adverse effect of an admission may be difficult to reverse. In addition, a lawyer may be able to make useful recommendations on the handling of any remedial efforts to ensure that such efforts are handled confidentially and in such a manner that the owner's interests are not compromised.

Building owners are being forced by the threat of liability to understand indoor air quality issues. By learning more about the means of legally protecting themselves against such liability, owners will be better prepared to address this emerging issue of the 1990s.

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THE UNIVERSITY OF TULSA
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The Center for Environmental Research and Technology

Psychosocial and Environmental Influences on Sick Building Syndrome

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PSYCHOSOCIAL AND ENVIRONMENTAL INFLUENCES ON "SICK" BUILDING SYNDROME

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Cornell University, Ithaca, NY 14853-4401

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"A physical symptom is a perception, feeling, or even belief about the state of our body."
(J. Pennebaker, *The psychology of physical symptoms*, 1982)

"(In the foundry)...they tipped the ladles to pour, white-hot metal splashed like pancake batter and sparks flew through the air.
'Do they do that job all day?' Robyn asked.
'All day, every day.' (Wilcox replied).
'It must be frightfully hot work.'
'Not so bad in winter. But in summer...the temperature can go up to a hundred and twenty Farenheit down there.'
'Surely they could refuse to work in conditions like that?'
'They could. The office staff start whingeing if it gets above eighty. But those two are men.' Wilcox gave this noun a solemn emphasis.
(D. Lodge, *Nice work*, 1989)

"A person who is aware of a given symptom may either not report it or may overreport it, depending on potential reinforcement or punishment from others."
(J. Pennebaker, *The psychology of physical symptoms*, 1982)

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SICK BUILDING SYNDROME

Researchers categorize indoor air quality (IAQ) problems in buildings in a variety of ways. Generally, IAQ problems are divided into those of "Building Related Illness" (BRI) and those of "Sick Building Syndrome" (SBS). BRI describes incidents where workers have been exposed to known contaminants in indoor air (e.g. exposure to bioaerosols from contaminated humidifiers causing humidifier fever, or to airborne bacterial infections such as legionnaire's disease). In BRI incidents a minority of workers usually are affected and show objective clinical signs of illness. Remedial action involves both treatment of cases (affected workers), and removal and control of the contaminant source (Bardana, Montanaro and O'Hollaren, 1988).

SBS is different. Cases typically show no clinical signs of illness; symptoms are vague; symptom prevalence is high (e.g. up to 80% of workers may report one or more symptoms), and complaints are chronic (Wilson and Hedge, 1987). SBS symptoms, as defined by the World Health Organization (1983), describes a general malaise which include symptoms of headache, lethargy, irritated nose and throat, eye problems, skin irritation (see Figure 1). Most SBS symptoms cannot be objectively measured, and there is high variability in symptoms among cases. SBS symptoms are thought to be associated with building occupancy because they often get better when the affected worker leaves the building. SBS symptoms are not thought to be life threatening.

SBS symptoms

Frequent

↑

Lethargy, tiredness, fatigue
Headache
Eye, nose, throat irritation
Cold/flu-like symptoms
Respiratory problems
Nausea, weakness, dizziness
Skin problems

↓

Infrequent

Figure 1 SBS symptoms defined by the W.H.O. (1983)

Buildings with a high prevalence of SBS cases often are called "sick" buildings, although again there is no consensus on how to gauge prevalence and what the criteria should be to discriminate between "sick" and "healthy" buildings?

The W.H.O. suggest distinguishing that "sick" buildings may fall into two categories:

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temporarily "sick" buildings - where there is an acute outbreak of health problems in either newly constructed or recently remodelled spaces. Symptoms typically dissipate over time (e.g., usually within 6 months symptom reports have ceased). Volatile compounds from building materials and finishes (e.g. paints, furniture, finishes, etc.) are the suspected cause of many of these complaints.

permanently "sick" buildings - where symptoms may persist over many years. Complaints are difficult to resolve because significant concentrations of indoor air pollutants cannot be detected, yet inadequate IAQ is suspected as the cause because symptoms are alleviated when away from the workplace.

Regrettably, the W.H.O. report did not define SBS in terms of the number or pattern of symptoms which might indicate an SBS case, the severity of symptoms, or the frequency of occurrence of symptoms for a case. Their report gave no explicit guidance on how to measure symptoms, over what time period, or even what symptoms should be measured. The W.H.O. did, however, acknowledge the possible role of psychosocial variables in the etiology of the syndrome.

The term "sick building" is now widely used to emotively describe certain places, although there remains no consensus on what constitutes either an SBS case or a "sick building". Despite this precarious research foundation, reports of SBS/IAQ problems have increased dramatically throughout the 1980s. Between 1971 and December 1978, the Hazards Evaluations and Technical Assistance Branch of the U.S. National Institute for Occupational Safety and Health (NIOSH) investigated only 6 buildings in which IAQ problems were suspected, whereas between 1978 and 1988, 523 such investigations were requested (426 of the total investigations were in office buildings). NIOSH suggested that problems generally seemed to be closely associated with energy conservation practices which includes the design, maintenance, and operation of building ventilation systems, and they suggested that inadequate ventilation (i.e. insufficient outdoor air, poor air mixing, poor air distribution, extremes of temperature and/or humidity, and filter maintenance problems), may account for the majority of SBS problems (Seitz, 1989). However, thorough analysis of the cause(s) of symptoms does not seem to have been undertaken there are no follow-up results by which to gauge the success of either their diagnoses or their recommendations.

METHODOLOGICAL ISSUES IN SBS INVESTIGATIONS

Most investigations of SBS use questionnaire surveys of building occupants to gauge the prevalence of symptoms and IAQ complaints. Usually, the questionnaires are self-

administered, although some studies have used interviewer administered instruments (Finnegan et al., 1987). A recent review of a number of the questionnaires used by SBS/IAQ researchers in the U.S.A., Canada, U.K., Denmark, and Finland found great variability in the symptoms under investigation, the timescale, the question wording, and the response scales used (Hedge, 1990). The extent to which this lack of standardization of either instruments or methods is confounding the results of SBS studies and hampering progress in elucidating the etiology of SBS is unknown.

Because most SBS studies use self-report questionnaires or interviews to collect symptom data, the results will be influenced by numerous psychological factors, including a worker's expectations, awareness of somatic symptoms and his or her tendency to attribute these to the environment. Data from poorly designed questionnaires are confounded by problems of recall bias and response scale bias. Recall bias describes those factors which affect the accuracy of our memory for events. In a study of prescribed drug use, Mitchell et al. (1986) showed that recall was significantly higher when patients were given specific drug names in questions rather than asked to name the drugs being used, even though all patients were on similar drug regimes. Response scale bias results from poorly designed response scales. Categorical scales with scale points of "often", "always", "sometimes", etc., present both the participant and researcher with considerable ambiguity (e.g., how often is often?). Apart from a draft ASTM standard which is currently under revision , there is a dearth of adequate guidance on how to design an SBS questionnaire and conduct a survey of SBS complaints in buildings.

PSYCHOSOCIAL INFLUENCES ON SBS REPORTS

Mass psychogenic illness

Mass psychogenic illness (MPI) describes "the collective occurrence of a set of physical symptoms and related beliefs among two or more individuals in the absence of any identifiable pathogen" (Colligan and Murphy, 1982). The dynamics of MPI depend on two process:

> **contagion** - the spread of affect or behavior from person to person in a group, where each case serves as the stimulus to be imitated by others.
>
> **convergence** - the simultaneous development of common affect or behavior among group members

Contagion and convergence processes often are triggered by an environmental event (e.g.,

a malodor. In the absence of an identifiable cause this trigger facilitates the expression of symptoms which individuals attribute to an environmental cause (e.g., a "mystery bug"). Symptoms of MPI usually include those of headache, nausea, weakness, dizziness, sleepiness, hyperventilation, fainting, and vomiting, and occasionally include a variety of skin disorders and burning sensations in the throat and eyes (Colligan and Murphy, 1982; Olkinoura, 1984; Boxer, 1985, 1990).

MPI symptoms probably are not purely psychogenic in origin, but are the result of interactions between pre-exiting stressful physical environment and work conditions (e.g., poor ventilation, poor lighting, excessive noise, tedious work, poor organizational climate, poor labor-management relations) and predispositions among individuals (e.g., gender, anxiety level), with a triggering event (e.g., malodor) and consequent psychosocial processes (e.g., management response to the perceived threat). The typical sequence of events from studies of MPI incidents is summarized in Figure 2.

Cognitive influences on SBS symptom reports

We do not possess the necessary physiological receptors to sense many indoor environmental variables. Many gases are colorless and odorless, we cannot detect airborne bacteria, we cannot directly see illumination or hear decibels, we cannot detect radiation, etc. We are imperfect devices for assessing physical environmental conditions in a building. Likewise, we are imperfect at sensing our actual bodily status. Because of these limitations, we often choose hypotheses to explain what we believe to be the environmental conditions and our corresponding somatic status. If, for example, we believe that the office ventilation is poor and that the air contains a colorless, odorless, yet noxious pollutant which causes eye irritation, we will likely behave accordingly by selectively monitoring our eye sensations for confirmatory sensory information. We may even rub our eyes more frequently thereby unconsciously creating this information. Such behaviors are quite common. When people are told to think about how itchy their nose is, many will eventually scratch their nose. When people are told to think about insects such as mites and fleas, many will eventually scratch their bodies or complain of sensations of itching skin. When people at concerts hear others coughing in the intermission, they are more likely to feel the urge to cough. When we read about diseases, we often begin to believe that we have the symptoms[1]. And so on.

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[1] Studies of medical students have shown that 70% of first-year students believe they have symptoms of diseases being studied (Pennebaker,1982).

Figure 2 Typical sequence of events in MPI incidents (adapted from Olkinoura, 1984)

Generalized Belief
(e.g. belief in presence of contaminant in a building)

↓

Precipitating Event
(e.g. perception of malodor or observation of usual behavior)

↓

Threat
(e.g. fear of pulmonary distress or illness)

↓

Physiological Arousal
(e.g. increased anxiety, agitation, excitement, fear, hyperventilation)

↓

Revised and Self-focussed Belief
(e.g. attribution of current and prior bodily sensations to the building)

↓

Social Contagion
(rapid spread of symptoms, legitimization of symptoms)

In an office, workers usually cannot precisely attribute causality to their symptoms (e.g., if a worker reports a headache s/he decide may have difficulty deciding whether this is caused by the IAQ, the lighting, the use of a VDT, the pressure of work, the noise, etc.).

SBS symptoms are percepts, and reports of these symptoms are affected by the same cognitive processes which influence all other aspects of perception (Pennebaker, 1982). Instructional set influences symptoms reports via its effect on attentional bias. Reports of nasal congestion are significantly affected by whether people are instructed to focus their attention on nasal congestion, which increases their reports of nasal stuffiness, or to focus their attention on free breathing, which decreases reports of nasal congestion under the

same environmental conditions (Pennebaker and Skelton, 1981). Symptom labels like "shortness of breath" convey different meanings to people, some interpret this as meaning slow, labored breathing, while others interpret this as rapid, shallow breathing (Pennebaker, 1982).

Similar processes may also influence our perceptions of indoor climate conditions. For men there is no significant correlation between measured relative humidity and perceptions of dry air, whereas there is a significant correlation between measured relative humidity and perceptions of dry air for women, although this is the reverse of that expected and reports of dryness increases with increasing relative humidity (Göthe et al., 1987).

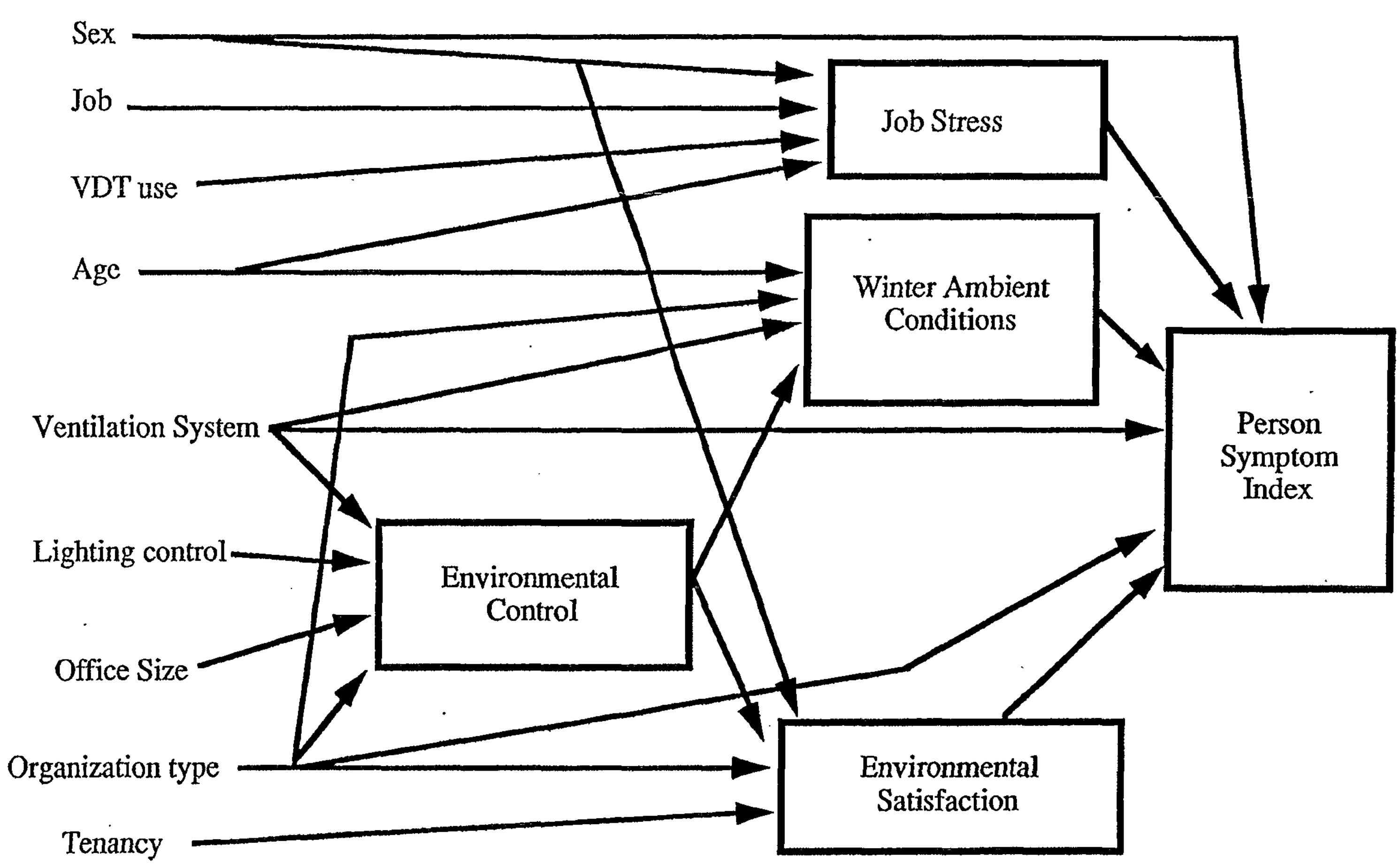
Worker's decisions in describing their symptoms and in attributing causality can be affected by any or all of the above influences, and by other psychological factors (e.g., moods, attitudes, beliefs) known to affect the workings of cognitive processes. Recent research on environmental illness among people with multiple chemical sensitivities suggests that their symptoms also can be explained by one or more commonly recognized psychiatric disorders, such as mood disorders, affective disorders, and anxiety disorders (Black, Rathe, and Goldstein, 1990).

A Model of Environmental Influences on Health in Offices

Hedge (1989) has attempted to capture something of this multi-factorial perspective in a descriptive model (see Figure 3). This model proposes that IAQ complaints and SBS symptom reports arise from the effects of *direct environmental* variables (e.g. exposure to pollutants), *indirect environmental* variables (e.g. worker's satisfaction with thermal conditions), and *non-environmental* variables (e.g. occupational variables such as job stress, VDT use, and individual variables such as gender, stress reactivity, etc.). It is suggested that, at any time, all of the factors described in this model can interact to change the **total stress load** on a worker, and this may either change the individual's sensitivity to environmental irritants or directly precipitate SBS symptom reports. The model suggests that many decisions about SBS symptoms are affected by discrepancies in how internal and external conditions are interpreted with respect to each other, and how individuals are able to cope with the total stress load.

This model also treats indoor conditions as an environmental subsystem which describes the inter-relationships between environmental services (ventilation system, lighting system) and ambient conditions (IAQ, thermal conditions, noise, and vibration). The environmental subsystem interacts with the building subsystem, which includes the characteristics of the building shell , the materials, furnishings and finishes may off gas pollutants, the office

Figure 4 Path model of sick building syndrome (adapted from Hedge et al., 1989b)



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layout, etc. The building subsystem will also affects workers' perceptions of environmental quality, their patterns of working, and workspace preferences (e.g. many office workers prefer private, enclosed offices to open-plan office layouts). Higher levels of distraction open-plan offices also may be stressful to workers (Dick et al., 1981).

The work subsystem also influences the environmental subsystem, because the activities of office workers directly affect IAQ. Breathing generates carbon dioxide, tobacco smoking releases pollutants and particulates, carbonless copy paper may release volatile organic compounds, laser printers may generate ozone, etc. Work activities will also influence the building subsystem (e.g. meetings in open office areas may be a bothersome noise source for proximate, uninvolved workers).

In conclusion this model suggests that whenever complaints and symptoms cannot be attributed to direct exposure to indoor air pollutants or indoor environmental conditions, symptom reports will be depend on individual discrepancies between perceived external states and the person's perceived internal state.

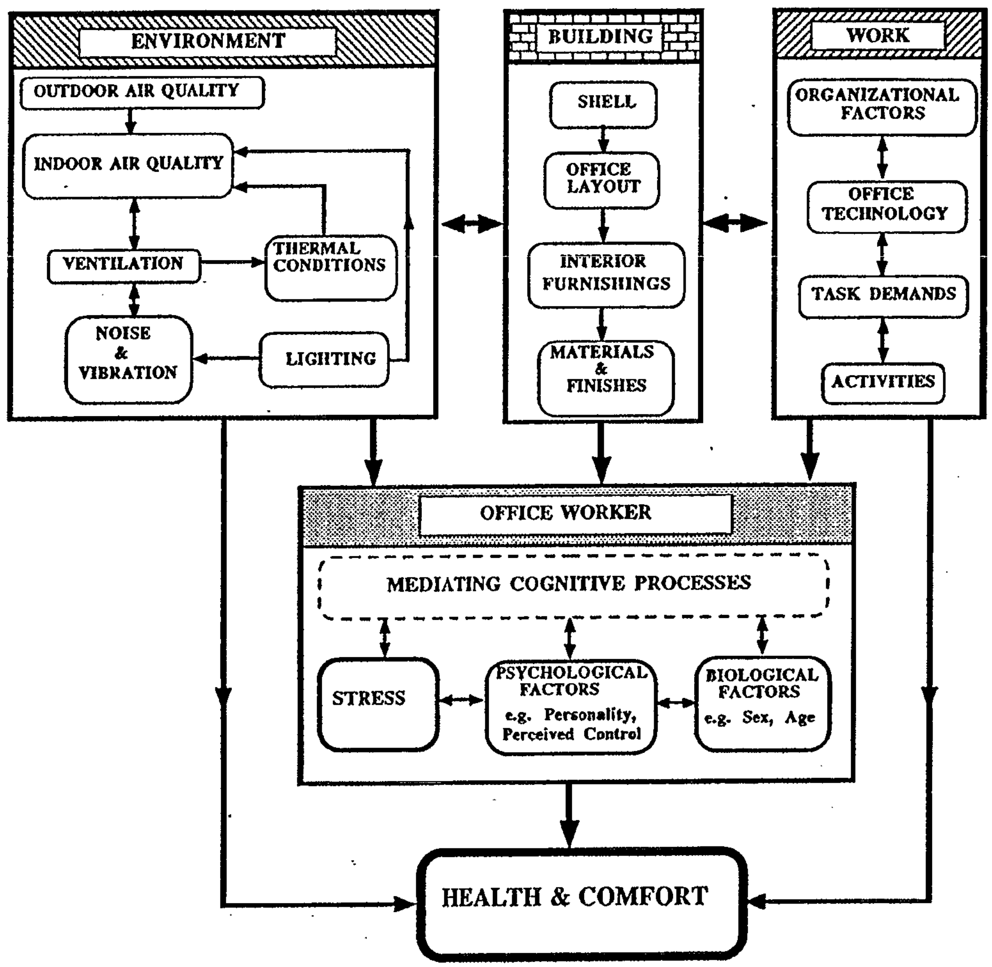
EVIDENCE THAT PERSONAL, PSYCHOLOGICAL, AND OCCUPATIONAL FACTORS AFFECT SBS

Several studies in different countries have found that SBS symptoms are significantly more prevalent in the air-conditioned than naturally ventilated offices (Hedge, 1984; Robertson et al., 1985; Burge et al., 1987; Hedge et al., 1989a,b; Mendell, 1990; Zweers et al., 1990). But apart from this finding there has been comparatively little agreement on the environmental cause of SBS and there is remarkably little research linking SBS symptoms to actual exposures to gaseous indoor air pollutants.

Rather, studies are beginning to give support to the model which has been described and to the usefulness of the concept of total stress load in understanding the etiology of SBS. An investigation of 3 offices found that although workers associated their symptoms with the environmental conditions, there was no correlation between day-to-day fluctuations in temperature and humidity, and reported symptoms of dry nose/nasal congestion. There was, however, a significant relationship between reports of SBS symptoms and levels of stress (Morris and Hawkins, 1987).

A number of studies have shown that SBS symptoms are more prevalent among women than men (Hedge, 1984; Robertson et al., 1985; Burge et al., 1987; Hedge et al., 1989a,b), and why this should be is unknown. There is conflicting evidence that other factors, such as age, atopy and allergy, and smoking status affect SBS symptoms, and

Figure 3 Systems model of the office environment (Hedge, 1989)



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whether or not these factors can account for the gender differences.

Occupational factors (job level, hours of computer use, job stress, job satisfaction, handling of carbonless copy paper, photocopying), psychological factors (perceptions of control, perceptions of ambient conditions, perceptions of comfort), and organizational factors (public sector versus. private sector buildings), significantly influence the prevalence of SBS symptoms among office workers (Hedge, 1988; Hedge et al., 1989b; Skov et al., 1989; Hedge, Erickson and Rubin, 1990). A path model describing some of these associations has been tested (see Figure 4).

CONCLUSIONS

The argument advanced in this paper is that the problems of SBS may not be simply a direct consequence of exposure to poor IAQ but rather the results of the combined effects of a variety of environmental and non-environmental factors. It is suggested that, much as we view many diseases as the outcome of multi-factorial processes, SBS might usefully be viewed as the outcome of a set of **multiple risk factors** which act to place a **total stress load** on a person at any time. But as yet we have only a poor grasp of what these factors might be and how they might affect each person.

Regrettably, progress on elucidating the etiology of SBS has been hampered by poor methodology. Many studies of SBS have used poorly designed questionnaires which are either biased, ambiguous, badly scaled, or conceptually ill-conceived. Moreover, questionnaires usually collect data on workers' perceptions of environmental conditions and health over extended periods of time e.g. one month, 3 months, 1 year, whereas measures of environmental conditions seldom are taken over such extensive periods, nor are they normally taken for each individual location in a building. So it is perhaps not surprising that little association between self-reported symptoms and IAQ has been found.

Most studies of SBS have failed to show significant associations between levels of air pollutants and SBS symptoms, but it is possible that a "missing" pollutant or pollutants is responsible for symptoms, and several recent research studies have suggested that the contents of office dust may be the most likely candidate for this missing link (Hodgson and Collopy, 1989; Armstrong, Sheretz, and Llewellyn, 1989; Leinster et al., 1990). However, in all studies which have measured non-environmental variables there is considerable evidence that personal, psychological, and occupational variables also affect reports of SBS problems. Although this remains a neglected area of study, future research should include investigation of at least the variables which have been described.

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THE UNIVERSITY OF TULSA
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Bioaerosols

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A DISEASE-RELATED APPROACH TO BIOAEROSOLS

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I. What are bioaerosols?
   A. Microorganisms shed by occupants and accumulating in air to unacceptable levels

   B. Microorganisms growing on building substrates and entering the air

   C. Effluents of non-human building occupants

II. Organisms producing bioaerosols
   A. Viruses
      1. Obligate pathogens
         a. Require a living host
         b. Do not grow on artificial substrates

      2. Very small; penetrate most filters

      3. "Simple"; protein coat + nucleic acid

   B. Bacteria
      1. Most are facultative saprophytes
         a. Able to grow outside of living tissue
         b. Many can grow on environmental substrates

      2. Small, usually unicellular, no organized nucleus

      3. Types of bacteria
         a. Gram negative rods
            (1). Environmental sources
            (2). Produce endotoxin as part of cell wall

         b. Gram positive rods
            (1). Environmental and human source
            (2). Some produce endospores

         c. Gram positive cocci
            (1). Human sources
            (2). Usually do not cause airborne disease

         d. Gram positive "mycelial" (actinomycetes)
            (1). Environmental sources
            (2). Produce dry airborne spores

   C. Fungi
      1. Nearly all are saprophytes and occupy environmental reservoirs

      2. Large size range from about 1 um to large, macroscopic fruiting bodies.

3. Types
    a. Unicellular forms (yeasts)
    b. Molds and mildews
    c. Plant disease organisms
    d. Mushrooms

4. Complex cell structure and metabolism
    a. Eukaryotic (like human cells)
    b. Multicellular (hyphae, mycelium, tissues)
    c. Specialized spores for dispersal
    d. Secondary metabolites (mycotoxins)

D. Arthropods
1. Mites
    a. Microscopic (about 100um)
    b. Free living in dust
    c. Fecal particles become airborne

2. Cockroaches
    a. Macroscopic pests
    b. Free living in damp places
    c. Fecal particles, body parts become airborne

E. Birds
1. Human choice exposure
2. Serum, droppings, ?dander airborne

F. Mammals
1. Rats, mice
    a. Pests or laboratory exposure
    b. Urine becomes airborne

2. Dogs, cats
    a. Human choice or laboratory exposure
    b. Saliva, urine become airborne

III. Diseases
A. Contagious disease
1. Infections
    a. Organisms must be alive
    b. Organisms do not cause other diseases

2. Human to human transmission

3. Agents
    a. Viruses
        (1). Examples: Influenza, measles, chicken pox, some colds

        (2). Never grow on inanimate substrates; may survive passage through ventilation systems

        (3). Very small, may not be removed by filtration

    b. Bacteria

(1). Examples: Tuberculosis

(2). Not usually growing on building substrates but may survive on surfaces

(3). Do not grow readily in culture

4. Risk factors
   a. Lack of specific immunity
   b. Dose
   c. Virulence of organism

5. Evaluation
   a. Disease identification/epidemiology
   b. Air and/or source samples not useful

6. Prevention
   a. Vaccination
   b. Avoidance/isolation
   c. Ventilation

B. Environmental-source virulent infections
   1. Type of disease: Infection
      a. Organisms must be alive
      b. Organisms do not cause other diseases

   2. Transmission mode: transmitted from outdoor reservoirs or housed animals

   3. Agents
      a. Rickettsia (Q-fever)
      b. Bacteria (anthrax)
      c. Fungi (Histoplasmosis, Blastomycosis, Coccidioidomyco.

   4. Risk factors
      a. Lack of specific immunity
      b. Dose
      c. Virulence of the organism

   5. Evaluation
      a. Disease identification/epidemiology
      b. Sampling usually not useful

   6. Prevention
      a. Avoidance
      b. Removal of reservoirs

C. Environmental-source opportunistic infections
   1. Type of disease: infection
      a. Organisms must be alive
      b. Organisms may also cause allergic/toxic disease

   2. Transmission mode:
      a. Outdoor reservoirs: cooling towers, compost

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b. Indoor reservoirs: water reservoirs, surface growth

3. Agents
    a. Bacteria
        (1). Examples: legionellosis, *Pseudomonas* pneumonia
        (2). Sources: water reservoirs

    b. Fungi
        (1). Examples: cryptococcosis, aspergillosis
        (2). Sources: accumulations of organic material; surface growth on semi-dry substrates

4. Risk factors
    a. Immune system malfunctions
        (1). Disease (AIDS, cancer, etc.)
        (2). Medications (steroids, immunosuppressants)
        (3). Substance "abuse" (smoking, alcohol)

    b. Dose

    c. Virulence

5. Evaluation
    a. Disease identification; epidemiology
    b. Source sampling of potential reservoirs
    c. Air sampling possible for *Aspergillus*

6. Prevention
    a. Removal of reservoirs
    b. Avoidance

D. Hypersensitivity diseases: Asthma, Hay fever
1. Immune response disease: exposure units do not need to be alive to cause a response.

2. Transmitted from outdoor and indoor reservoirs

3. Agents
    a. Fungi (any fungus; ex. *Alternaria, Aspergillus*)

    b. Pollen (air dispersed; ex. grass, ragweed, many trees)

    c. Arthropods (especially indoor pests; ex. mites, cockroaches.

    d. Mammals (especially indoor pets, laboratory animals; ex. dogs, cats, mice, rats)

4. Risk factors
    a. Genetic

    b. Exposure patterns (differ for development of sensitization and development of symptoms)

    c. Antigenicity

5. Evaluation
   a. Disease identification

   b. Environmental evaluation
      (1) Observation
      (2) Source sampling
      (3) Air sampling

6. Prevention
   a. Avoidance
   b. Remove reservoirs
   c. Desensitization

E. Hypersensitivity diseases: Hypersensitivity pneumonitis

1. Immune response; agents of exposure need not be alive to elicit effect.

2. Transmitted from indoor reservoirs

3. Agents
   a. Bacteria (*Bacillus*, thermophilic actinomycetes)

   b. Fungi (any small-spored fungus, or fungus growing in a water reservoir where antigens can be eluted and sprayed into the air as small droplets; ex. *Penicillium, Cladosporium*).

   c. Birds (serum, droppings)

4. Risk factors
   a. Host risk factors unknown

   b. Exposure levels and patterns

   c. Adjuvants (either intrinsic as in thermophilic actinomycetes, or endotoxin)

   d. Antigenicity

5. Evaluation
   a. Disease identification

   b. Environmental evaluation
      (1). Observation
      (2). Source samples
      (3). Air sampling

6. Prevention
   a. Avoidance
   b. Remove environmental reservoirs

F. Toxicoses

1. Direct cellular toxic effect; exposure units need not be alive.

2. Transmitted from environmental reservoirs either in association with source organisms or as inanimate droplets containing the active metabolite.

3. Agents
    a. Bacteria (Endotoxins, exotoxins)

    b. Fungi (Mycotoxins; ex. *Aspergillus flavus, Stachybotrys atra, Fusarium sporotrichoides*)

4. Risk factors
    a. Direct cellular toxic effect; human dose response is constant

    b. Toxicity

    c. Dose

5. Evaluation
    a. Disease identification

    b. Environmental evaluation
        (1). Observation
        (2). Source sampling to identify toxigenic organisms
        (3). Air sampling to verify airborne exposure

6. Prevention
    a. Avoidance
    b. Removal of environmental reservoirs

G. Airborne microbial contamination that probably does not cause disease
1. Agents include human-source bacteria (e.g., *Micrococcus, Staphylococcus epidermidis, Streptococcus salivarius)*

2. Grow readily in culture and constitute the usually-measured bacterial flora on indoor air samples.

3. Accumulate when per person ventilation is inadequate

H. Sick building syndrome
1. Symptoms include: headache, dizziness, nausea, eye irritation, lethargy, chest tightness, sinus congestion

2. Hypothetical causes include: inadequate outdoor air ventilation which causes accumulation of volatile organic compounds, fungal and/or bacterial toxins, other biogenic odors, comfort and stress factors.

3. Risk: unknown

4. Evaluation:
    a. Observation
    b. Measurements (ventilation)
    c. Sampling (CO_2, etc.)

IV. General approach to building investigations involving bioaerosols

A. Do symptoms match a microbially induced disease? If not, check for adequate ventilation or other kinds of contaminants.

B. Is the epidemic human to human transmission problem? If so, check for adequate per person fresh air.

C. Is the disease legionellosis? (diagnosed, serotyped)

1. Is there more than one case? Remember: a single case does not necessarily implicate the work environment. Most *Legionella* exposure probably occurs in the home.

2. Look for source of aerosolized contaminated water; culture and compare serotypes.

a. Air sampling is not effective

b. *Legionella* is a common organism. Finding it does not imply disease or even risk.

D. Is there an obvious microbial contamination problem?

1. Walk through and look for water problems, obvious mold growth, or microbial slimes; musty or locker-room odors; damp fabric (carpet, drapes, furniture).

2. Check air intake location for possible outdoor sources (e.g., compost piles, cooling towers, construction)

3. Obvious microbial problems should generally be remedied without sampling. Regardless of sampling results you will recommend remediation.

E. General protocol for microbial sampling

1. Use the ACGIH Guidelines

2. Source samples can document the presence of contamination in suspected reservoirs, and the nature of the organisms. Examine samples visually and microscopically, and culture with 24 hours on definitive media for fungi, bacteria and actinomycetes at appropriate temperatures.

3. Use both cultural and particulate methods for air sampling

4. Use good quality volumetric devices; never rely on gravity collections.

5. Data analysis

a. There are no published numeric guidelines

b. Acceptable levels depend on the sampling method

c. Acceptable levels depend on the organism

(1). Thermophilic actinomycetes should not be present

(2). *Stachybotrys atra* should not be present.

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(3). Pattern of taxon prevalence should parallel that in outdoor air for the season.

d. "Grab" samples may seriously misrepresent bioaerosol status of an environment.

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Division of Continuing Education
The Center for Environmental Research and Technology

Building Performance Relative to Bioaerosols and VOCs

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3.2 ENERGY EFFICIENT HVAC

3.2.2 INTERNAL HVAC POLLUTION

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1 - LARGE BUILDINGS

1.1 OUTDOOR AIR INTAKES / EXHAUSTS

External sources of contaminants such as bioaerosols and combustion products often enter HVAC system outdoor air inlets. Contaminants in exhaust emissions often entrain in outdoor air inlets. HVAC then becomes **direct** source of air contaminants.

Remediation

- Outdoor air intakes should be located at a site (preferably on roof) where the ambient air quality is the best. This means that grade level sites should be avoided. Outdoor air inlets should be located at sites so that possible entrainment of contaminants from cooling towers, exhaust and relief vents, and other contaminant sources including those from other buildings is avoided;
- Fences should be used to separate outdoor air intakes from exhaust and relief air vents;
- Research is needed on airflow patterns around buildings in relation to optimal location or outdoor air inlets and exhausts;
- Keep outdoor air intake plenums clean;

1.2 ACCESSIBILITY OF INTERIORS OF CENTRAL AIR HANDLING UNITS (AHUS) FOR CLEANING

Accessibility to interiors of AHUs is often poor. Dirt, debris accumulate. Fleece factor becomes important. Result: HVAC-AHU becomes a direct source of air contaminants.

- Ceiling AHUs, rooftop AHUs, and central system AHUs must be designed for easy access for cleaning;
- Access panels should have smooth inner surfaces;

Research

- What is adequate access? Size of access panels? How many? Locations? How can penetration and leakage problems be minimized? Minimum guidelines of acceptability for manufacturers are needed.

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1.3 MIXING PLENUMS OF AHUS

Assume AHU is accessible. Plenums (and rest of AHU) often are poorly maintained. Pesticides, VOCs, microbials often present in plenums. HVAC then becomes a direct source of air contaminants because of poor maintenance.

Remediation

- Keep mixing plenums clean;
- The use of odor maskers is unethical;
- The use of pesticides and the presence of open drains should be prohibited in mixing plenums.

Research

- Maintenance protocols are needed for rooftop units, central AHUs, AHUs in ceiling plenums;
- Quantify effects of maintenance deficiencies by panel of judges and by measurement of air contaminants;
- Develop maintenance rating checklists (scales) so that inspector can quickly rate this aspect of HVAC performance.

1.4 FILTER BANK OF AHU

Filter dirt likely acts as secondary emission source for ETS and VOCs. Moisture in filters means microbial amplification. All indoor air even in well maintained system passes through dust cake. This is probably not good for people.

Remediation

- Change filters frequently;
- Keep them dry.

Research

- Develop new technology so that filter dust cake in HVAC system air stream is avoided;
- Sensory perception. Can panel of judges "sense" dust cake?
- What is maximum air velocity in HVAC that is compatible with prevention of water droplet entrainment in air stream?
- Consensus protocols for filter maintenance for acceptable IAQ should be developed.

1.5 HEAT EXCHANGERS IN CENTRAL AHU

Contaminants arise in heat exchanger plenum because of stagnant water (biofilm) in drain pans, humidifiers and water spray systems and because of use of microbiocidal agents, etc. In some systems, cooling tower water (***Legionella***) is in cooling coil. HVAC system becomes a **direct** source of air contaminants.

Remediation

- Allow no stagnant water in HVAC; access to the drain pan for cleaning is essential;
- Coils with direct water connection to cooling towers must never leak (especially in hospitals);
- Biocides that can be aerosolized into indoor air should not be used in operating AHUs (drain pans), Water spray systems, and Humidifiers.

Research

- Detailed protocols for heat exchanger maintenance should be developed;
- Develop new types of humidifier that do **not** aerosolize microbiologicals, biocides, and corrosion inhibitors.

1.6 POROUS INSULATION IN HVAC

Porous insulation is found in AHUs (housing interior surfaces), supply air plenums, VAV boxes. Dirt and debris deposit over time in pores. Under high moisture conditions (for example downstream of operating cooling deck coils) fungi and bacteria will amplify of nutrients (dirt). Insulation also likely acts as secondary emission sources for VOCs and ETS.

Remediation

- Vacuum clean; replace insulation; externalize insulation; replace AHU; place insulation between metal surfaces.

Research

- What are acoustical penalties associated with externalization of insulation?
- Determine alternatives to cooling coil for latent heat removal; this might include desiccant dehumidification of incoming outdoor air;
- Determine if there are other approaches for dampening fan noise in HVAC.

1.7 COMMON RETURN AIR PLENUM

Common return air plenum can be source (direct and indirect) for entry of air contaminants into HVAC. Wet insulation and ceiling tiles are sources of microbials. Insulation can be secondary emission source of VOCs. Loose insulation can be source of irritating fibers in occupied space. Maintenance of AHUs in plenum is poor because of location (AHU then becomes source of air contaminants).

Recommendations

- Ducted returns should be used in place of common return plenums;
- Avoid the use of materials that can not be cleaned in common return air plenums; avoid the use of cellulose; avoid the use of high surface area materials.

Research

- Compare the economics of use of common return air plenum versus ducted return in view of IAQ, productivity, and overall construction costs;
- Carry out sensory panel tests to compare acceptability of ducted versus plenum return-air buildings;
- Research is needed on cleanable and non adsorbant/absorbant materials for use in common return plenums.

1.8 PERIPHERAL HVAC SYSTEM (INDUCTION UNITS; FAN COIL UNITS; UNIT VENTILATORS)

Peripheral units are associated with IAQ complaints (S. Burge). These units are excellent **direct** sources of microbials and secondary emissions of VOCs and ETS (especially hotels). Maintenance is almost always poor.

Recommendations

- Maintenance is required;
- Alternatively, these units should not be used in HVAC if maintenance is impossible.

Research

- Determine if these units be maintained in cost effective manner **or** should their use be discouraged (use central system AHUs instead);
- Quantify sensory and contaminants effects associated with the use of these units.

1.9 HVAC AS INDIRECT SOURCE OF AIR CONTAMINANTS

Providing outdoor air to breathing zone is an expensive and inefficient method for controlling some air contaminants such as VOCs, ETS, and human-shed microorganisms.

Recommendations

- Use source control.

Research (Human-shed microorganisms)

- Sick people (common cold) should stay away from work (source control). Health care plans should emphasize illness prevention. Study sick time loss in building with and without common-cold-management policies.

1.10 PRESSURIZATION OF BUILDINGS: EFFECT OF HVAC

Air contaminant from outdoor locations around a building can enter the indoor air because of improper pressurization. Air contaminants within a building can migrate from zone to zone because of pressure differentials. Thus, HVAC can become an **indirect** source of air contaminants.

Recommendations

- Total make-up air supply and total exhaust (relief) air output must be considered at **all** times in order to maintain proper pressurization;
- Strong external and internal contaminant sources must be excluded from the building or restricted by control at source in order to prevent building-wide dissemination.

1.11 CONCLUSIONS

HVAC systems can be major sources of indoor pollutants. Location of contaminants sources can be determined qualitatively by visual preassessment. Quantification of source strengths and health effects is difficult.

Remedial actions are often very obvious:

- Improvement in maintenance is most often associated with remedial actions;
- Some HVAC-sourced contamination sources however can be remediated only with great difficulty (for example, porous insulation; relocation of poorly located outdoor air inlets).

2 - RESIDENCES-MECHANICAL VENTILATION

2.1 MOISTURE - RESIDENCES

The presence of water in ventilation systems and in the occupied space can lead to significant indoor pollution (esp. microbial).

Recommandations

- Prevent accumulation of water/water vapour in ventilation systems;
- Prevent condensation of moisture on walls, windows, in the building envelope, parts of structure that communicate with the occupied space.

Research

- Better insulation systems (that avoid thermal bridging)for building envelopes are needed.

2.2 MAINTENANCE OF RESIDENTIAL VENTILATION SYSTEMS

All mechanical ventilation systems can become inefficient in terms of energy use if they are not properly maintained. Dirt and debris can accumulate in poorly maintained systems and can become primary and secondary sources of contaminant emissions.

Recommendations

- Keep ventilation systems clean by regular maintenance;
- The use of odor maskers is unwise and unethical.

Research

- Research on protocols for filter maintenance is needed;
- Develop audit procedures to access maintenance status.

2.3 MATERIAL SELECTION FOR VENTILATION SYSTEMS

Some materials used in ventilation systems can become primary (e.g., caulks, tapes) and secondary (porous insulation) sources of contaminant emissions.

Recommendations

- Internal surfaces in forced air systems should be smooth and should not be conducive substrate for mould growth;
- Locate fan motors outside of ventilation air stream;
- Use materials which have low source strenghts of potential contaminant emissions.

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2.4 PRESSURIZATION

Some air contaminants enter residential indoor air because of improper pressurization differentials.

Recommendations

- Make-up air must be provided in such a manner that contaminants such as radon and combustion products (from the soil and garages, respectively), are not drawn into the occupied spaces;
- The movement of air contaminants from one dwelling (apartment) to another (through walls) must be prevented by proper zoning or by provision of separate ventilation systems.

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IRSST

Pilot Study on Indoor Air Quality

NATO OTAN

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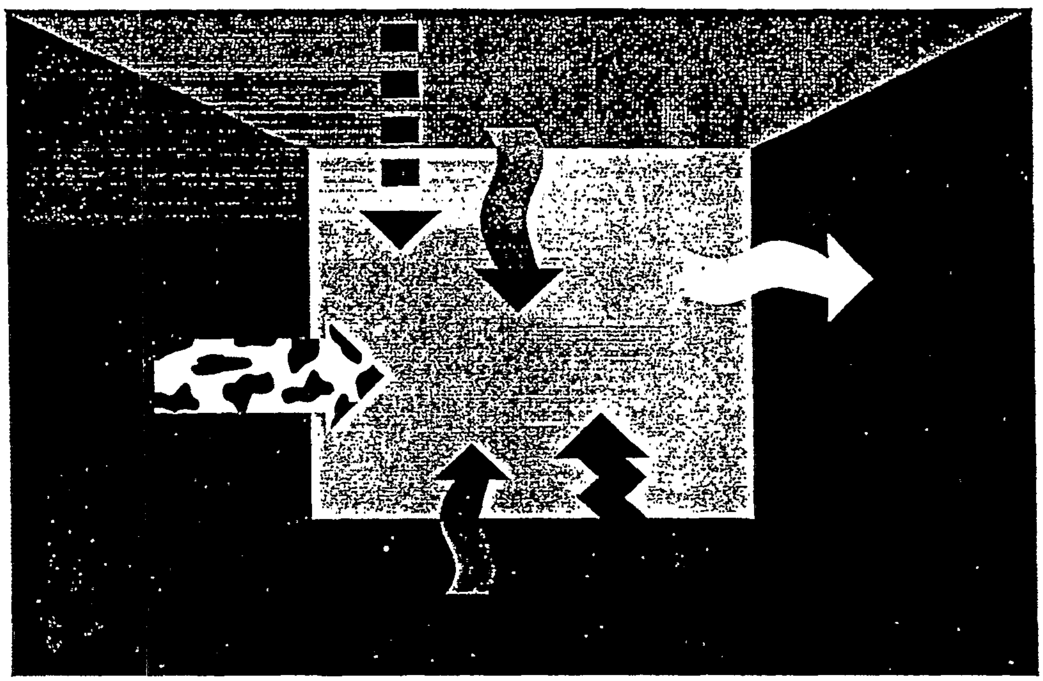

Report on a Meeting Held in Sainte-Adèle, Québec,
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RANK ORDER ASSESSMENT OF VOLATILE ORGANIC COMPOUNDS IN INDOOR AIR QUALITY EVALUATIONS

by

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RANK ORDER ASSESSMENT OF VOLATILE ORGANIC COMPOUNDS IN INDOOR AIR QUALITY EVALUATIONS

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ABSTRACT

Rank order assessment is presented as a method for evaluating airborne volatile organic compounds (VOCs) present in nonindustrial indoor environments. VOCs are collected on Tenax and analyzed by gas chromatography/mass spectrometry. In this method, specific VOCs are ranked in decreasing order of abundance for an indoor site and this list is then compared to the concentrations of the same analytes in the outdoor air. It may be obvious at this point if the indoor VOCs are atypical and possibly contributing to indoor air quality (IAQ) problems. When total indoor and outdoor VOC concentrations are similar, rank order assessment is helpful in determining sources of specific VOCs found in indoor air.

INTRODUCTION

Various procedures have been proposed to assess the effects of VOCs on the occupants of nonindustrial indoor environments. It has been suggested that a total concentration of VOCs between 160 and 5,000 micrograms per cubic meter ($\mu g/m^3$) may be indicative of a threshold associated with annoyance and discomfort among sensitive occupants (1).

Total concentrations of VOCs in indoor air are typically 2 to 10 times higher than those outdoors (2,3). Problem buildings are likely those with higher total indoor/outdoor concentration ratios (3). A number of investigators have called attention to the specific nature of various VOCs, including those emitted from new construction and finishing materials, cleaning materials, combustion processes, etc. (2,3,4).

We have performed sampling for VOCs in 43 buildings with IAQ problems. The objective of this study is to illustrate that rank order assessment is useful in deciding if the indoor VOCs are atypical and possibly contributing to IAQ problems.

MATERIALS AND METHODS

We have performed IAQ evaluations in 105 buildings during the past 2 years. In 43 of these buildings, air sampling for VOCs was performed in complainant zones, often in noncomplaint zones, and always in the outdoor air as a reference (usually on the roof of the building). Complaints that elicited VOC sampling usually included odor annoyance and irritation of the

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eyes, nose, and throat. VOC analytical results presented in tables are from one representative VOC sample from a complainant zone and from one outdoor air (reference) VOC sample in each building.

Sampling for VOCs was performed by drawing air at a flowrate of 50 to 75 cc/minute through Tenax for 5 to 8 hours. Tenax was thermally desorbed and analyzed by gas chromatography/ mass spectrometry (USEPA T01 Method).

Analytical results of VOC sampling were evaluated by rank order assessment. In this method, concentrations of individual analytes at an indoor site are listed in descending order of abundance, and this list is compared to the concentrations of the sample analytes in the outdoor air. Comparison of the indoor and outdoor lists of analytes offers a simple method for assessing sources of indoor VOCs. This method has been used to assist in the interpretation of sampling data for airborne saprophytic fungi and bacteria (5).

RESULTS AND DISCUSSION

Case studies are presented to illustrate the use of rank order assessment for evaluation of VOC sampling results.

Building A. Complaints of inability to concentrate, earaches, and muscle aches were reported by employees in a hospital laboratory in a suburban location. The total concentration of VOCs both indoors and outdoors was approximately 500 $\mu g/m^3$ (Table 1).

The VOCs outdoors were dominated by methylene chloride, possibly due to ongoing paving of a nearby parking lot. Indoor VOCs were dominated by C_9H_{12} alkyl benzenes and xylene and ethyl benzene, most likely from solvents used in the laboratory. Local exhaust ventilation was recommended for removal of indoor aromatic hydrocarbons.

Table 1

Specific VOCs ($\mu g/m^3$) in Building A

	Indoors	Outdoors
C_9H_{12} Alkyl benzenes	200	6
Xylene and ethyl benzene	80	2
Methylene chloride	40	400
1,1,1-Trichloroethane	20	60
Total VOCs	447	543

Building B. Air sampling was performed in tenant offices in an urban high rise, 30-year-old office building on several occasions. The air handling units serving the tenant offices also serve (and recirculate air from) a total of 25 floors. Renovation work was ongoing on non-tenant occupied floors following asbestos removal.

Concentrations of total VOCs were determined during the autumn and winter (Table 2). When air handling units were providing minimum (<15 percent) outdoor air (winter) to occupied spaces, indoor VOC concentrations in some areas reached 265 $\mu g/m^3$ and exceeded outdoor

air levels by a factor of at least 10. Indoor concentrations under minimum outdoor air ventilation conditions were consistently dominated by aliphatic hydrocarbons, most likely due to emissions from the new furnishings (modular office partitions and carpets) being installed on other floors.

On autumn days when the air handling units were providing maximum (up to 100 percent) outdoor air (economizer mode of operation of air handling units), total indoor VOC concentrations were in the 75 to 125 $\mu g/m^3$ range, and rank ordering showed that the indoor VOCs were not dominated by any single type of compound.

Table 2

Specific VOCs ($\mu g/m^3$) in Building B

	Indoors	Outdoors
Maximum Outdoor Air (autumn day)		
C_6-C_{12} Aliphatics	12	15
C_9H_{12} Alkyl benzenes	10	5
Xylene and ethyl benzene	9	2
Total VOCs	81	48
Minimum Outdoor Air (winter day)		
C_{13} and Greater Aliphatics	70	<LOC*
C_6-C_{12} Aliphatics	47	2
1,1,1-Trichloroethane	20	0.2
Total VOCs	263	16

* LOD means limit of detection.

Building C. Employees in a large 1-year-old, 6-story office building in a suburban location began complaining of headaches, eye irritation, nausea, and fatigue soon after the building was occupied. Outdoor concentrations of total VOCs were approximately 50 $\mu g/m^3$. Indoor total VOC concentrations ranged from 400 to 2,500 $\mu g/m^3$, for an indoor/outdoor ratio between 8 and 50 (Table 3).

Rank order assessment indicated indoor VOCs were dominated by components of solvents (2-butanone, up to 2,000 $\mu g/m^3$) and emissions from furnishings, new construction materials (aliphatic hydrocarbons and toluene), cleaning compounds (limonene), and deodorizers (dichlorobenzene), in concentrations up to 100 times greater than those outdoors.

Examination of the heating, ventilating, and air-conditioning (HVAC) system revealed that it had been inadequately balanced (variable air volume terminals had minimum set points of zero) and was providing inadequate amounts of outdoor air (less than 10 cubic feet per minute per person on average).

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Table 3

Specific VOCs ($\mu g/m^3$) in Building C

	Indoors	Outdoors
2-Butanone	800	20
C_6-C_9 Aliphatics	80	2
1,1,1-Trichloroethane	75	<LOD
Methylene chloride	45	6
Total VOCs	1,237	55

Building D. Employees in a 5-year-old public building in a suburban location complained of headaches, fatigue, eye irritation, and inability to concentrate. Total VOC concentrations outdoors were approximately 15 $\mu g/m^3$, with indoor/outdoor ratios in the 20 to 50 range (Table 4).

Examination of analytical results revealed that concentrations of aliphatic hydrocarbons were up to 115 times greater indoors than outdoors. Examination of the HVAC system operation records revealed that inadequate amounts of outdoor air (variable air volume terminals had minimum outdoor air set points of zero) had been provided during the 5 years of occupancy. Although we could not exclude the possibility that elevated concentrations of VOCs were due to internal sources such as the use of cleaning chemicals, it was speculated that high indoor VOC concentrations were associated with emissions of VOCs from furnishings and finishing materials used during building construction.

Table 4

Specific VOCs ($\mu g/m^3$) in Building D

	Indoors	Outdoors
C_6-C_{12} Aliphatics	345	<LOD
1,1,2-Trichloroethene	33	<LOD
2-Butanone	8	<LOD
Benzaldehyde	<LOD	4
Total VOCs	432	15

Building E. Sampling for VOCs was performed in an urban 40-story office tower. The indoor/outdoor concentration ratio was approximately 10 (Table 5). Rank order assessment showed that Freon-113, used in refrigeration systems and by some laser printers, was present in concentrations nearly 200 times greater than outdoors. Other possible sources of indoor VOCs included solvents used in an unexhausted printing area and cleaning agents.

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Table 5

Specific VOCs ($\mu g/m^3$) in Building E

	Indoors	Outdoors
Freon-113	187	1
1,1,2-Trichloroethene	124	1
C_6-C_9 Aliphatics	43	8
$C_{10}H_{14}$ Alkyl benzenes	26	8
Total VOCs	508	45

CONCLUSIONS

Sampling for VOCs was carried out in 43 buildings. Rank order assessment of analytical results was used (limited data presented from each of five buildings) to help determine if indoor VOCs were atypical, especially with regard to those in the outdoor air. Complainant areas in 26 of the 43 buildings were characterized by atypical VOC profiles such as those illustrated for Buildings A-E.

Rank order assessment provides a useful method for evaluating VOCs on a building-by-building basis. In Building A, comparison of total indoor and outdoor concentrations of VOCs would not have indicated the differing indoor and external sources of specific VOCs. In IAQ studies, rank order assessment of VOCs is a much more appropriate problem solving approach than simple comparison of total VOC concentrations.

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BAYER

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Operation and Maintenance and Its Effect on Healthy Buildings

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OPERATION AND MAINTENANCE PRACTICES FOR MAINTAINING OPTIMUM INDOOR AIR QUALITY

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The quality of the indoor air to which occupants are exposed, particularly in the office environment, has become of increasing concern during this last decade. Various investigative studies into "sick" and "problem" buildings indicate that a significant percentage of building occupants are exposed to indoor environmental conditions which may result in adverse health effects, occupant discomfort, and reduced employee performance and productivity. The results of numerous indoor air quality investigations by researchers of the Georgia Tech Research Institute (GTRI) and others have shown that design inadequacies and poor building operation and maintenance (O&M) practices are the primary categories which dominate sources of air quality problems.

Frequently poor O&M practices are the result of improperly installed and inaccessible HVAC systems and ductwork, lack of training of facility maintenance and management staff, cost restrictions, lack of understanding of the importance of maintenance practices, improper use of chemicals and cleaners, or attempts at energy conservation. Design inadequacies can also be the cause of poor O&M practices. O&M practices are defined here as applying to all aspects of building operation and maintenance including HVAC, custodial services, renovation procedures, pest control practices, smoking policies, etc.

The cost of poor indoor air quality can be quite costly for employers. A conservative estimate for annual salary costs is $200 per square foot. The typical annual average office building heating/cooling costs are about $2 per square foot. Salaries are the most significant of all operating costs in almost every company. Poor O&M practices which result in decreasing indoor air quality can be counterproductive to employee and tenant productivity.

HOUSEKEEPING AND CLEANLINESS

Cleanliness and housekeeping of the building and mechanical-ventilation areas are extremely important. Dust should be removed daily, not just stirred around. Soap and water should be used in bathrooms and food preparation, storage, and eating areas in place of highly perfumed cleaners. The use of aerosol sprays should be minimized. The storage of chemicals in the area used for return and outside ventilation air can result in contamination of the occupied areas with these chemicals. The use of highly perfumed products and deodorizers can trigger hypersensitivity reactions in building occupants. GTRI researchers have investigated several different incidents where the use of highly perfumed products have triggered hypersensitivity reactions.

Case 1: In one study, the occupants were complaining of headaches, nausea, respiratory problems, and odors. The problems had increased significantly in the month prior to the investigation. The fourth floor, the floor with the most complaints, had a strong sickenly-sweet odor. It was discovered that the maintenance staff had sprayed a vanilla-scented deodorizer on the primary HVAC system filters when they replaced the filters. They sprayed the deodorizer on the filters in response to occupant complaint of foul, rotten garbage-like odors permeating throughout the fourth floor. Inspection of the building air intakes revealed that six sewer roof vents were located directly in front of the building air intake. Strong sewer gas odors were emanating from the vents and the odorous air was being drawn into the building ventilation system and distributed throughout the occupied zones. This design inadequacy resulted in poor O&M practices resulting in human responses of discomfort.

Case 2: The emissions from continuous-feed bathroom deodorizers resulted in occupant complaints of respiratory problems, eye irritation, nausea, headaches, and lethargia. The office areas were filled with foam-filled modular office partitions. The partitions emitted strong odors of the grape-scented bathroom deodorizer used in the buildings. It was found that the bathrooms exhaust was vented to the top of the elevator shafts. When the elevator operated it created a vacuum in the bathroom

drawing bathroom deodorizer emissions into the elevator shafts and distributing it throughout the building. The foam-filled modular office partitions served as sinks for the bathroom deodorizer emissions and then became secondary sources of the contaminants. The result was that over time high levels of the deodorizer contaminants existed in the occupied zones and caused irritation effects among the occupants. As in Case 1, design inadequacies combined with poor O&M practices resulted in human discomfort complaints.

PEST CONTROL

Improper application of pest control has been resulted in a plethora of homeowners complaints. The frequent improper application of chlordane was one of the major contributing factors to its being banned. Pest control should be applied during periods of inoccupation with increased ventilation and according to the manufacturers' directions. Building occupants should be aware of the hazards of personal use of pesticides.

Case 3: The occupants of a business office sought medical help after suffering chemical burns in their respiratory systems and in their mouths, having headaches, lethargia, and other symptoms of poor indoor air quality. The office was in a larger office complex. The occupants of the business office were the only people in the complex suffering symptoms. Investigation into the building revealed that there was poor ventilation efficiency in the space, but nothing in the building design indicated this the cause of the sudden onset of severe symptoms although the area had a chemical smell. Upon questioning of the occupants, it was learned that they had been spraying the space regularly, several times a day on and around their desks with pesticides. They also said that the weekend before they became so sick that they had released two bug bombs. The office complex turned off the ventilation system on the weekends as an energy conservation measure. When the occupants returned for work on Monday morning, the pesticides were still in the air since there had been no ventilation of the space to remove the contaminants. The business office residents did not follow proper usage guides for pesticides.

HVAC SYSTEMS

Inadequate design of HVAC systems frequently results in poor O&M practices. Inaccessibility is one of the most common poor design features encountered.

Case 4: Complaints of headaches, lethargia, and confusion were being expressed by the occupants of a combination emergency medical facility and office building facility. The building had three roof-top ventilators were only accessible by climbing a steep ladder located in an extremely small, crowded storage room. The door to the roof was a heavy trap door which could only be opened and closed by precariously hanging from the ladder. The maintenance personnel had removed the filters from the roof-top systems rather than risk life and limb negotiating the roof access. As a result, the condensate pans clogged and water leaked into the supply ductwork and the ceiling below. Ductboard was used for supply ducts. The building had a plenum return which was fiberglass insulated on top of the ceiling tiles instead of the plenum ceiling. Insulating on top of the ceiling tiles resulted in an extremely hot and humid plenum. Therefore, when cold air-conditioned air escaped through unsealed areas in the ductboard seams, condensation on nearby surfaces occurred. This condensate dripped onto the ceiling insulation providing conditions for microbial growth. The facility manager replaced ceiling tiles when they disintegrated, but did not replace the fiberglass insulation. No effort was made to investigate the source of the condensation by the facility manager. The inaccessibility of the roof-top ventilators and the poor design and construction of the complex resulted in poor O&M practices and subsequent microbial contamination of the occupied zones of the building.

Case 5: Complaints were being received by the facility managers by the tenants on the ninth floor of a twelve story office building. The occupants of the Northwest wing of the building were suffering eye irritation, sinus infections, and sneezing and coughing fits. The occupants also complained of a dirty, musty smell which was particularly strong early in the morning. The majority of the suffers had been diagnosed by physicians as having allergic reactions to molds. The occupants most severely afflicted were not the typical of those usually suffering reactions to poor indoor air quality. All of the victims were young, healthy, athletic individuals

who had not previously suffered from allergic type reactions. The person who was most severely afflicted was a mountain climber. During the investigation, microbial swab samples of the ninth floor air handling condensate pan, coil, outdoor air intake, and selected diffusers. Standing water was present in the ninth floor air handler. The analysis of the swab samples indicated fungal growth on the outside air intake, bacterial growth within the condensate pan and coil, and no growth on the diffusers. The genera which were detected are known to cause allergic reactions. Poor O&M practices probably resulted in airborne microbial contamination and the resultant allergic reactions among the building occupants. It was recommended that the facility maintenance personnel clean the various areas of the HVAC system with dilute chlorine bleach and hot water.

Case 6: In another investigation, the tenants of an office building were suffering suspected indoor air related symptoms of headaches, eye irritation, and respiratory system irritation. The tenant was a brokerage firm which did not employ chemical usage, had a single copier, and had no other obvious sources of occupant generated contaminants. This was the only area in the office complex which reported occupant symptoms. The most severely afflicted occupant reported that he could smell cigar smoke when the adjacent tenants used the conference room next to his office. These neighboring tenant was a photographic and graphics arts firm. It was discovered that in this area there were several large color and black-and-white copying systems, automatic darkroom systems, and other wet-processing copy systems. None of these systems was properly vented to prevent contamination of the recirculated air. In addition it was found that there was a break in the firewall around the building perimeter which allowed direct transference of the contaminants generated in the graphic arts firm into the brokerage firm. The poor construction of the firewall, deviation of space use from the design specifications, and lack of proper exhaustion of contaminants being generated by the processes of the graphic arts firms resulted in poor indoor air quality and occupant discomfort complaints.

RENOVATION PRACTICES

Renovation should be conducted during periods of non-occupation and with increased ventilation. Proper commissioning of new and renovated space should be

followed. All renovation materials should be properly stored in sealed containers.

Case 7: A corporate senior vice president suddenly began suffering anaphylactic shock for three successive days within five minutes of entering his office. After three trips to the hospital, he instructed the building maintenance staff to find the problem with his office. Investigation showed that major renovation was being conducted on the entire floor two floors below the VP's office. On this floor, immediately below the VP's office, the wallpaper paste, paints, caulking compounds, and waste buckets were being stored. The waste buckets and several of the wallpaper paste buckets were being left open. A pathway was discovered for contaminant transference from this floor into the VP's office. The supplies were removed to a proper storage area and were sealed. Once this was done, the VP was able to enter his office without going into shock.

SUMMARY

Inadequate design and poor O&M practices have resulted in a seemingly large majority indoor air quality related complaints. Inadequate design frequently results in poor O&M practices. Other causes of poor O&M are laziness, lack of training and understanding, improper use of chemicals and cleaners, cost restrictions, and attempts at energy conservation. Every facility should have established O&M guidelines and policies which are designed to assure optimum indoor air quality. These guidelines should set up a schedule for O&M, renovation practices, crisis response, tenant and occupant interaction practices, provide adequate instructions and training, and must importantly the guidelines should be implemented.

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Carbon Monoxide Exposure in Mountaineers on Denali

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ABSTRACT

Carbon monoxide (CO) poisoning is a particular hazard for persons camping in cold, stormy, high altitude locations such as Mt. McKinley (Denali). Recently, two climbers were killed by CO on Denali while cooking in a tightly sealed tent. CO has also contributed to high altitude illness. To assess exposure in various shelters we measured CO levels with a portable monitor in a mountaineering tent, igloos and snow caves during an ascent of Denali.

Concentrations measured at altitudes between 2000 and 5200m usually exceeded the U.S. Environmental Protection Agency's 1-hour standard of 35ppm, reaching a mean value as high as 165ppm in a snow cave. Based on the data from this preliminary study, the use of a larger vent hole, approximately the size of a ski pole basket, appears to provide enough ventilation to maintain CO levels at more acceptable levels in snow caves and igloos.

INTRODUCTION

Ever since the early polar explorers began using portable kerosene stoves to cook and melt snow for water, numerous accounts of people being poisoned by the exhaust gases from such stoves have been published (1–3). One such account comes from the journey of Stefansson to the North Pole. He reported that while using a Primus stove (a small kerosene stove) in a snow house, the walls "became iced and impervious to gases," whereupon two of his companions collapsed in the house while he and the fourth member of the party managed to extinguish the stove and escape before collapsing: "An hour later three of the party members were well enough to go into the house again..." (1).

In the modern era, we have the advantage of considerable research on topics such as emission rates of carbon monoxide (CO) from stoves and lanterns (2,4), the concentrations of CO that can be found in tents and snow houses (1–5), and the amount of carboxyhemoglobin (COHb) found in the blood after carbon monoxide exposure (3). The acute health effects of exposure to carbon monoxide are well established (6). Most recently, Cohen (4), using a combination of laboratory work and computer modeling predicted that high levels of CO could be attained in tents when a cooking stove was used even for periods less than 30 minutes if ventilation were inadequate.

Despite considerable knowledge in this area, morbidity and mortality from CO poisoning persists on Mt. McKinley (Denali). In 1986, two Swiss climbers died on Denali while cooking in a tightly sealed tent at 4400m, ironically, only a few meters from the medical research camp. The purpose of this study was to better define the exposure to CO on Denali under typical mountaineering conditions in typical shelters and to test various methods of reducing the hazard by changing shelter ventilation.

Measurements were made during and after the use of the stove for meal preparation. The data indicate that CO exposure may reach toxic levels in all types of shelter and that adequate ventilation in snow shelters can be established by larger vent holes.

METHODS

Two of the authors (WT and SM) made the measurements during a climb of Denali in June of 1985. Measurements were made in three types of shelters used for mountaineering: tent, igloo, and snow cave (Figure 1). Because climbers often encounter temperatures well below freezing and strong winds, these shelters are designed to be small in volume and to have low ventilation rates. The tent was nylon, designed for two persons, with double-wall construction and a vestibule for cooking that was open to the inside of the tent. Igloos were made with cut snow blocks, and the cooking and sleeping level was higher than the entrance. Snow caves were also constructed with the living area higher than the entrance to help maintain warmth.

Carbon monoxide (CO) measurements were made using a calibrated CO monitor (Gas Tech Portable, Model CO-82). The instrument was zeroed before and after each series of measurements. A calibration standard of 180ppm CO was used at 90m, 2600m, 3400m, and 4300m above sea level to define a relationship be-

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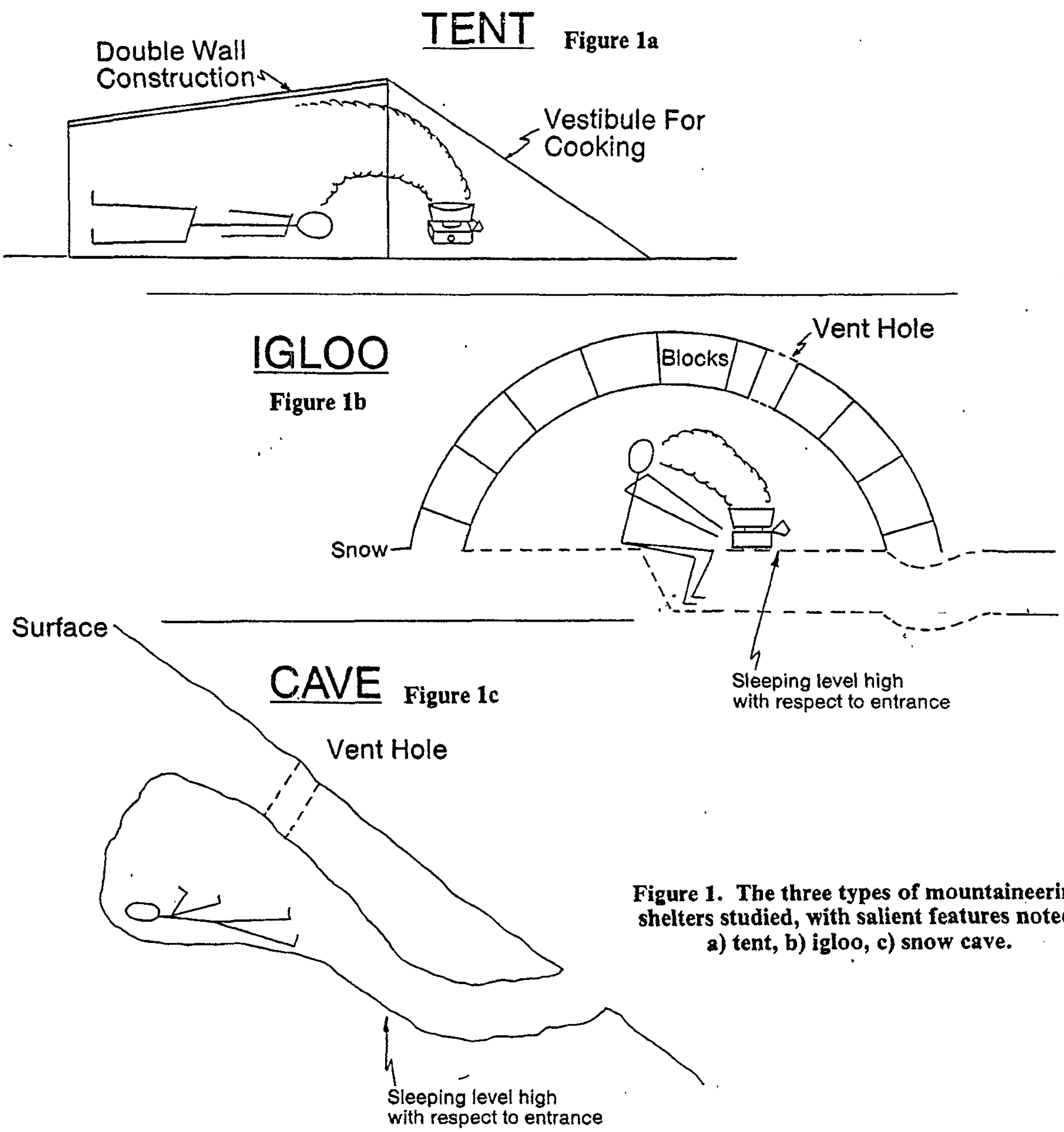

Figure 1. The three types of mountaineering shelters studied, with salient features noted: a) tent, b) igloo, c) snow cave.

tween the instrument's response to the standard and the altitude. When plotted, an altitude correction function was derived and applied to the data. (The instrument reads low at high altitude due to decreased partial pressure of gas.) This was preferred over adjusting the instrument at each altitude. Readings of the CO instrument were recorded at approximately 10-minute intervals during monitoring.

Three different stoves with similar fuel consumption rates were used during the testing. These were an Optimus 111B, an Optimus 8R, and a MSR Firefly. All stoves burned white gas and were in good operating condition. None had special jets for high altitude, and no attempts were made to alter the air/fuel ratios. The maximum fuel consumption rate for two of the three stoves (111B and 8R) has been measured to be approximately 210 grams/hour and is estimated to be less for the Firefly.

Measurements were made during the preparation of the meals in each of the dwellings. These periods typ-

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ically lasted between one and two hours and consumed 150–300 grams of fuel. The detector was located in the breathing zone of the occupants, approximately 1.5 to 2 meters horizontally from the cookstove and approximately 1 meter vertically, depending on the particular shelter. Efforts were made to standardize this sampling location. CO measurements were also made after the stove was turned off to calculate ventilation rates. Assuming exponential decay, the ventilation rate (air exchange rate of the dwelling) is the slope of the logarithm of CO concentration plotted against time. A more in-depth study of this method and its assumptions can be found in American Society of Testing and Materials (ASTM) (7). In snow caves, all measurements were repeated with different sized ventilation holes. Ventilation area was changed in the tent by partially opening or closing the zippered door. The reported CO concentrations have been corrected for altitude.

RESULTS

Tent

Table 1 gives the data that was taken inside the tent at 2300m using the 111B stove. During the last two runs, the CO concentrations were notably higher than the first two runs and were above the toxic level (50ppm). The outdoor wind speed during the first run was 7m/s, while there was no detectable wind during the following measurements. This explains the high ventilation rate in run 1 as measured by CO decay rate.

Igloo

The results from using the 111B and 8R stoves in the igloos at 2600 and 4300m are presented in Table 2. CO reached toxic levels in all tests when the stoves were burning for more than fifteen minutes. The first test was made while the snow blocks were still fresh and porous, which may have led to the relatively high air ventilation rate (air exchange rate) observed. The last three runs were made in igloos that had relatively less permeable walls due to glazing and accumulated snowpack. There was a decrease in ventilation rate for the later tests.

Snow Cave

Table 3 summarizes the results for the snow caves at 5000 and 5300m using the 8R and MSR stoves. While in these snow caves, the highest CO concentration was recorded (190ppm). Therefore, we investigated the relationship between concentration and the vent size.

Vent Area Versus Ventilation Rate

In a snow cave, vent holes of different sizes were made to test the relationship between air exchange rate and the size of the hole. The size of the vent area does not include the opening at the cave entrance, since the floor of the cave is higher than the entrance. Three basic sizes were tested: an ice axe shaft hole (approximately $10cm^2$), a ski basket hole (approximately $50cm^2$), and a hole made with the handle of a shovel (approximately $80cm^2$). The relationship between vent area and the ven-

Table 1

TENT

Stove Type	111B	111B	111B	111B
Altitude (meters)	2300	2300	2300	2300
Delta temp. (degrees C)	11	6	8	6
Wind Speed (m/s)	7	none	none	none
Burn Time (hrs)	.75	.5	.5	.5
CO conc (ppm)				
# Points	10	5	6	6
Max.	40	40	80	110
Mean	25	35	50	70
Min.	15	25	15	55
Gross Ventilation (cm^2)	4500	19400	2600	19400
Ventilation Rate (air exchange/hr)	150	65	13	38
# of points in decay curve	3	15	10	9

tilation (air exchange) rate is shown in Figure 2. In this limited data set the ventilation rate closely correlates with the gross vent area (R^2=0.903). A hole of 50cm² more than doubled the ventilation rate, and also kept CO concentrations below the EPA standard of 35ppm for one hour.

Air Exchange Versus Dwelling Type

Air exchange rates in the three shelters were determined by decay of CO over time with the source off. Figure 3 shows typical decay rates for the tent, the igloos, and the snow caves with holes. For the tent, the entrance door was half open. Even without perceptible wind, the ventilation rate was calculated at 65 air exchanges/hr (ACH). The inside vs outside temperature difference was about 6°C. CO concentrations reached background ambient values in three minutes. For the igloo, with a vent hole of approximately 45cm², a typical air exchange rate was five air changes/hour, and the CO concentration had not reached ambient levels after 30 minutes. For the cave with a vent hole of approximately 20cm², a typical air exchange rate was 3.6 air changes/hour, and after 45 minutes the CO concentration was still relatively high.

DISCUSSION

The observations and experiments performed on Denali between 2000m and 5200m were made under typical snow camping conditions. The stoves and shelters are all common to mountaineering. The snow burn

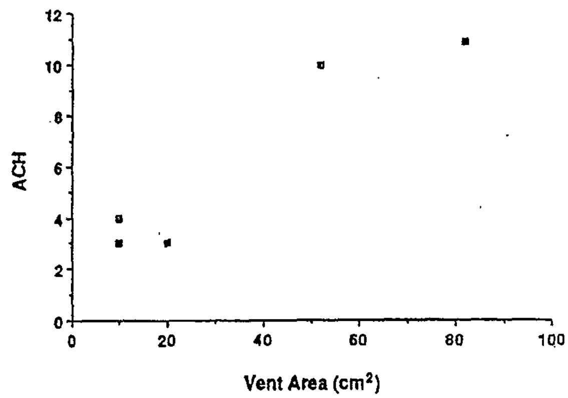

Figure 2. Air exchange rate (ACH) vs. size of vent area in a snow cave. A vent greater than 50 cm sq resulted in higher exchange rates.

times were actual meal or water preparation times required to support two or three climbers. It is important to note that 8 of the 13 sets of measurements of CO exceeded 50ppm during stove use. Our data, though limited, indicate that mountain climbers on Denali who cook in shelters may be at risk of acute carbon monoxide poisoning and that increasing shelter ventilation effectively reduces the risk.

Average CO concentration ranged from 15ppm to 165 ppm, varying primarily with shelter ventilation rates but also with fuel consumption and stove type. The air

Table 2

	IGLOO			
Stove Type	111B	111B	8R	8R
Altitude (meters)	2600	4300	4300	4300
Delta temp. (degrees C)	17	23	23	23
Wind Speed (m/s)				
Burn Time (hrs)	1	1	.5	.25
CO conc (ppm)				
# Points	8	14	5	4
Max.	85	140	80	40
Mean	80	120	70	35
Min.	70	80	60	30
Gross Ventilation (cm²)	none porous	45 glazed walls	45 glazed walls	45 glazed walls
Ventilation Rate (air exchange/hr)	13	5	4	14
# of points in decay curve	17	22	10	10

exchange rates were determined to be lowest in igloos and snow caves where the walls are more impervious to air flow and ventilation holes limited. We confirmed similar CO levels and ventilation rates during March 1986 in snow caves at 1000m and 1500m in the Presidential Range of the White Mountains of New Hampshire (unpublished observation).

The plot of the CO concentration decay suggests that the mechanism for ventilation in the snow caves and igloos is relatively similar, while the tent may differ. Wind is one factor expected to exert much more influence on ventilation in a tent compared to snow shelters, especially once an interior ice-glazed surface is formed. It should be noted that during the first experimental run inside of the tent, a wind of approximately 7m/s was blowing outside. This run resulted in an air exchange value of 150 per hour. Subsequent measurements were in calm conditions, and air exchange rates were closer to the other two shelters but still generally higher for the tent. The much larger vent area of the tent appears to be a distinct advantage. It should be noted that tightly sealed tents, however, could also form an ice glazed interior surface in the absence of wind or be covered with snow on the outside; even tents have the potential for low air exchange rates, as demonstrated by the recent deaths on Denali.

The effectiveness of using larger vent holes in snow caves is an important finding. We found the relationship between the air exchange rate in the snow cave and the vent area was relatively linear, implying only a small contribution to the ventilation rate was made by diffusion of air through the semi-porous snow and ice. In order to make a rough estimate as to what vent size would be required to keep the CO concentration below a certain level, the CO concentration was also plotted against the vent size. We recommend a minimum of $50cm^2$ vent in a snow cave, which seems to be adequate even if the snow walls are glazed.

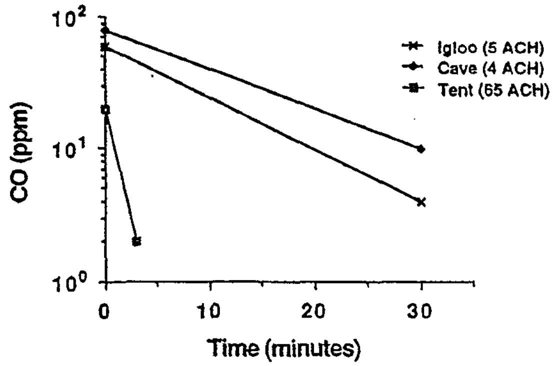

Figure 3. Carbon monoxide concentration in parts per million over time for the three different shelters. The shelter with high air exchange rate (ACH) had the most rapid decline in CO after the stove was turned off.

The emission rate (or source strength) of the cooking stove also influenced the level of CO. Based on fuel consumption rates determined for the 111B and 8R and boiling times for all three stoves, it appeared that the MSR Firefly had a lower fuel consumption rate and

Table 3

SNOW CAVE					
Stove Type	8R	8R	MSR	MSR	8R
Altitude (meters)	5000	5000	5000	5300	5300
Delta temp. (degrees C)	8	11	14	11	5
Wind Speed (m/s)					
Burn Time (hrs)	1 hr.	1	2	1	.25
CO conc (ppm)					
# Points	10	6	11	4	3
Max.	190	35	95	65	30
Mean	165	30	70	50	28
Min.	70	25	60	38	25
Gross Ventilation (cm^2)	11	52	20	11	81
Ventilation Rate (air exchange/hr)	4	10	3	3	11
# of points in decay curve	18	4	6	11	5

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thus possibly a lower emission rate.

The concentrations of CO recorded in the various shelters are high enough to produce significant toxicity. Of the 13 total experiments, eight showed mean concentrations of 50ppm or higher. Fifty ppm CO results in a carboxyhemoglobin level of about 8%, assuming the kinetics of uptake and equilibration remained the same as sea level. There is some evidence, however, that CO uptake is increased because of the hyperventilation induced by the hypoxia of high altitude (8,9). Although 8% COHb is not considered toxic at sea level, such a level will exacerbate the altitude hypoxia. The normal arterial oxygen saturation (SaO2%) of a climber on Denali after two days acclimatization to 4400m is 80%. A decrease in oxygen carrying capacity of 8% to an effective SaO2% of 72% due to COHb would result in a very significant drop in oxygen transport, rendering the climber at a "physiologically" higher altitude and could easily precipitate acute mountain sickness. In 1985, two Denali climbers required rescue because of severe acute mountain sickness induced by CO exposure (10). Low-level CO exposure may thus contribute to altitude illness. Further evidence for an additive effect of CO and altitude hypoxia was a study by Vollmer *et al.* (11). Four of seventeen healthy subjects at a simulated altitude of 4725m collapsed when COHb reached 9% to 19% (9), values not associated with collapse at sea-level. The EPA has more recently reviewed the combined effects of CO and altitude (12).

The higher CO concentrations recorded in our study are clearly in the range to cause CO toxicity, depending mostly on the exposure time. Symptoms of CO poisoning are identical to those of acute mountain sickness; i.e., headache, nausea, dizziness and lassitude. Therefore, differentiating the two may be impossible. Also, because of the lower inspired oxygen pressure, the rate of elimination of CO from hemoglobin may be impaired.

Two climbers found dead of CO poisoning from cooking in a tightly sealed tent on Denali in 1986 had COHb levels of 65.6% and 56.9% at autopsy, levels considered lethal even at sea level. Although the interactions of CO and altitude hypoxia need further investigation, it is clear that CO poisoning poses a serious risk when using stoves in shelters at high altitudes.

SPECIFIC RECOMMENDATIONS

As a result of the measurements obtained, the authors recommend that tents, igloos and snow caves always be vented when combustion appliances are used. We suggest that mountaineers adopt the practice of creating a vent hole in igloos and snow caves that is at least the diameter of a ski pole basket whenever a stove is in use, especially at high altitude. This vent should be located in the vicinity of stove operation and as high as possible in the dwelling. To maintain vital warmth offered by the shelter without risk of physical impairment from CO poisoning, the vent hole may be plugged with snow after finishing use of the stove. Using this method, the authors on this McKinley climb were able to maintain temperature within properly constructed igloos and caves in the range of 15 to 20° C above ambient except when the vent was open.

In light of the recent mountaineering deaths and illness attributable to CO poisoning and increasing activity of high altitude mountaineering, further research is needed in this area. Altitudes above 4000m are sufficiently hostile without the added danger of carbon monoxide.

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Low Flow Rate Sharp Cut Impactors for Indoor Air Sampling: Design and Calibration

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Two single round nozzle impactors have been developed for use in Harvard's indoor air pollution health study. Both impactors operate at flow rates of 4 L/m and are nearly identical, differing only in their cut sizes of 2.5 μm and 10 μm aerodynamic diameters. Two identical cascaded stages of the same cut size are used to obtain sharp cut-off characteristics. The particles are deposited on impaction plates made of oil impregnated, porous material to reduce particle bounce and are discarded. Only the particles collected on the afterfilter are analyzed. Special care has been taken to collect the particles uniformly on the afterfilter to aid in particle analysis.

The impactors were calibrated with a vibrating orifice monodisperse aerosol generator. However, due to the sharp cut of the impactors, doublets and triplets in the calibration aerosols, even in small quantities, gave erroneous calibration curves. Therefore, the number of doublets and triplets in the challenging aerosols were measured and appropriate corrections made to the calibration curves.

Inertial impactors are popular instruments for the collection and size classification of airborne particles. Their popularity stems from their ability to classify particles by aerodynamic diameter, an important parameter in aerosol studies, and for their ability to make sharp particle fractions (cuts) at precisely known diameters. This has resulted in impactors capable of collecting particles on sampling substrates with very specific and well defined particle size ranges.

When sampling particles for subsequent chemical/elemental analysis and possible association with human health effect, characterizing reliable size separation is important. Size fractionation of deposited particles occurs in the respiratory tract during inspiration. Further, physical and chemical processes result in bi- or tri-modal distribution of suspended particles in the atmosphere. Because alkaline particles tend to be greater than 3 μm in diameter and acidic particles tend to be less than 1 μm, a sharp size separation in this range would be desired to prevent neutralization of acidic aerosols collected on a filter. Further the distinct separation of particle mass by size permits source resolution by multivariate statistical analysis techniques using the elemental and chemical composition of the "fine fraction" particle mass. The Harvard Air Pollution Health Study has collected size fractionated ambient aerosols in each of the study cities since 1979 using virtual dichotomous impactors.[1]

For these biological and physical reasons, an impactor was developed for sampling airborne particles in the residential indoor environments. The impactor provides a sharp size separation at either 2.5 μm or 10 μm and results in uniform deposition of particles on filters (Teflon, quartz) selected for subsequent mass, chemical and/or elemental analysis. This paper describes the design and calibration of the particle impactor used in Harvard's Indoor Air Pollution Health study.[2]

Two versions of the impactor were developed; one with a particle cutsize of 2.5 μm aerodynamic diameter and the other with a cutsize of 10 μm, both at flow rates of 4 L/m. The particles collected on the impaction plates are discarded and not analyzed. The particles of interest, i.e. those less than the cut size, are uniformly deposited on afterfilters to be analyzed for mass and elemental composition.

Special care was taken in the design of the impactor to obtain as sharp a cut as possible at the desired particle size. This was done to provide comparability to the fine fraction particles collected with dichotomous samplers operated at central monitoring sites in each of the six communities. However, due to the sharp cut, the impactor was difficult to calibrate if the calibrating aerosols were not perfectly monodisperse. Calibrating with a vibrating orifice monodispersed aerosol generator (VOMAG, TSI, Inc. Model 3050, St. Paul, MN) gave erroneous results, due to the doublets and triplets in the challenging aerosol, even though they were low in number. Therefore, the number of multiple particles had to be measured and the impactor calibration corrected accordingly. This procedure is described in a later section.

Impactor Design

The design criteria for the impactor was rather simple; it had to provide a sharp classification at the design cutpoints of 2.5 or 10 μm aerodynamic diameter at a flow rate of 4 L/m and collect all penetrating particles uniformly on an afterfilter. For the design described here, the filter holder had to accommodate a 2 × 2-in. plastic filter slide containing a 37-mm diameter filter. However, any 37-mm filter could be used with a slight modification to the filter holder.

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A schematic diagram of the impactor is shown in Figure 1, and photographs of the impactor parts are shown in Figure 2. For either cut size, two identical cascade stages have been used for two reasons. First, it has been shown that the combined particle cutoff characteristic of two stages with the same cutsize is sharper than for a single stage.[3] Second, if any particles do bounce from the first stage due to overloading, they should be collected on the second stage with the net result being an impactor with very little, if any, particle bounce onto the afterfilter. To further reduce particle bounce, the impactor substrates are oil-impregnated porous metal plates.[4] In practice light mineral oil is used. The oil provides a sticky surface from which the particles will not easily bounce, and the oil will wick up through the deposit to continually provide a sticky surface to incoming particles. In this manner, overloading problems do not occur as long as the deposit does not become too close to the exit of the nozzle.

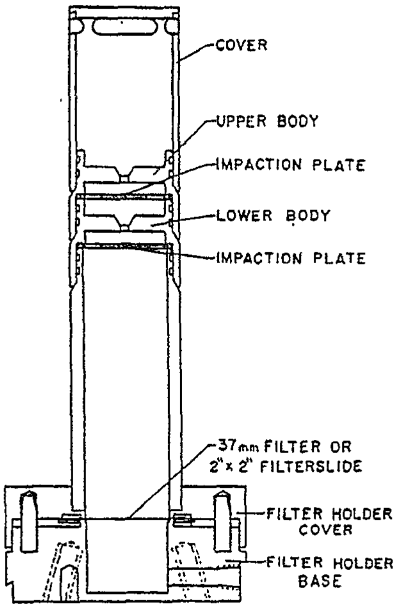

Figure 1. Schematic diagram of indoor air sampling impactor.

The filter holder consists of a base and cover that press a plastic filter slide between two gaskets. Two over-center draw latches hold the filter assembly together. The lower impaction plate sits directly on the tube extending above the filter holder and is held in place by the body containing the nozzle which slips onto the tube. The upper stage is identical to the lower stage and is assembled in the same manner. A cover slides onto the upper body to complete the impactor assembly. Important parameters of the impactor are given in Table 1. The impactor nozzle diameters were initially designed from theory[5] and then adjusted slightly after calibration to achieve the exact desired cut points.

Calibration

The impactors were calibrated using monodispersed liquid and solid particles generated with a VOMAG.[6] The liquid particles were oleic acid with a uranine dye tracer and the solid particles were ammonium fluorescein. The solid particles were used primarily in the impactor collection efficiency tests while the liquid particles were used in the particle loss studies. Both the uranine and ammonium fluorescein are fluorescent dyes. The use of ammonium fluorescein as a test aerosol and a description of its pertinent properties are described by Stober and Flachsbart[7] and Vanderpool and Rubow.[8] A fluorescent uranine dye tracer was added to the oleic acid to enable easy detection of where the particles are collected.[9] This is accomplished by passing monodispersed particles through the impactor and then washing the particles from various parts of the impactor with 0.001 N sodium hydroxide for the case of the liquid particles or 0.1 N ammonium hydroxide solution for the solid particles. The washes are then analyzed for fluorescent dye content with a fluorometer. By knowing the quantity of wash solution and the dye concentration in each wash, the relative amount of particles at all locations can be measured. In this manner, the collection efficiency curves and interstage losses can be determined.

Table I. Design parameters and cutoff diameters of the impactors in the MST indoor air sampler.

Nominal cutoff diameters. μm	2.5	10
Nozzle diameter, cm	0.244	0.620
S/W	2.0	0.86
Re	2140	900
Cutoff diameter, μm		
Theoretical	2.52	10.4
Experimental	2.52	9.8

When using the VOMAG, some doublets (particles with twice the volume as the particle size being generated), triplets, etc. are generated. Although the numbers of such multiplets may be small, their presence must be taken into consideration when using the dye tracer technique, since the quantity of dye in a doublet will give the indication of two particles of the generated size being collected rather than one particle with twice the volume. The impaction characteristics of singlets and doublets are obviously different.

The technique used to make corrections for multiplets was to monitor the number of particles of each size with a TSI Model 33 aerodynamic particle sizer (APS)[10] and to reduce the measured collection efficiency accordingly. To achieve the correction, the calibration is started with particles of size such that the multiplets are collected with 100 percent efficiency, and each subsequent calibration performed with slightly smaller particles. In this manner of developing the efficiency curve, the collection efficiency of the multiplets is always known and can be backed out of the calibration.

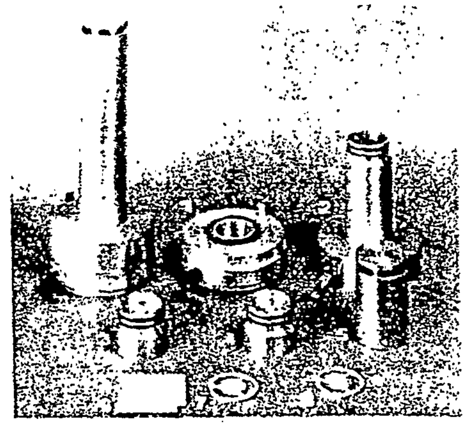

Figure 2. Indoor air sampling impactor: 1. Base and afterfilter holder; 2. Body with filter hold down clamps; 3 and 4. Nozzles; 5. Inlet; 6. Afterfilter in 2" × 2" filter slide; 7 and 8. Impaction plates; upper left: assembled impactor.

The measured efficiency is a function of the contribution of the singlets and each of the multiplet particle sizes. For a test aerosol consisting of primary (singlets), doublets and triplets, the measured efficiency, E_m, is given by

$$E_m = f_1E_1 + f_2E_2 + f_3E_3 \quad (1)$$

where f_1, f_2 and f_3 are the volume fractions of the singlets, doublets and triplets in the test aerosol and E_1, E_2 and E_3 are the actual particle collection efficiency for the singlets, doublets and triplets. The collection efficiency of the singlets, E_1, can be computed from Equation 2,

$$E_1 = \frac{E_m - f_2E_2 - f_3E_3}{f_1} \quad (2)$$

therefore, the solid line in Figure 3 and not the middle dashed line which would be obtained if no corrections were made. It is of interest to note that the *correction is only* important for the low values of efficiency and becomes very critical for impactors with sharp classification characteristics.

The amount of correction, of course, depends upon the fraction of particles that are multiplets. As shown for the above example in Figure 3, 5 percent multiplets substantially alter the collection efficiency curve for efficiencies less than 20 percent. If the number of multiplets drops to 2 percent, the correction is small, as shown by the curve labeled 1.8 percent doublets and 0.2 percent triplets in Figure 3. Conversely if the multiplets increase to 10 percent (also

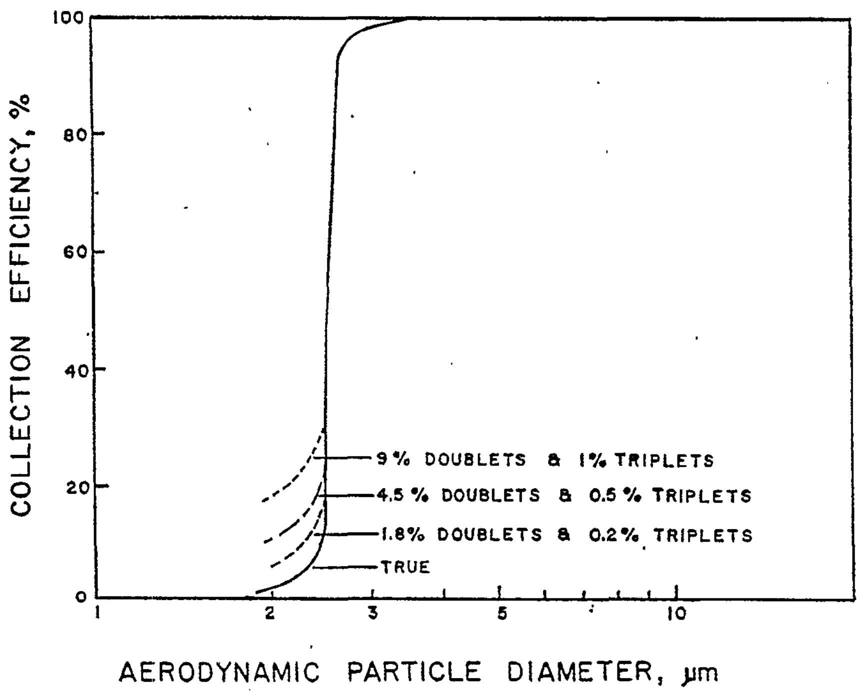

Figure 3. Effect of calibration aerosol doublets and triplets on experimentally determined efficiency curve.

providing the collection efficiency for the larger particle sizes and the volume fraction of each aerosol type are known.

For example, assume that a particle size of 2.1 μm aerodynamic diameter has been generated and 11.3 percent are collected on the filter and 88.7 percent on the impaction plate. Further, assume that there are, on a number basis, 4.5 and 0.5 percent doublets (2.65-μm diameter) and triplets (3.03-μm diameter) respectively. Without correcting for the multiplets, the calibration curve (middle curve in Figure 3) shows the 2.1-μm particles being collected with an 11.3 percent efficiency. However, the 2.65-μm diameter particles are collected at 95 percent efficiency, and the quantity of particles on the impaction plate should be reduced by 8.1 percent. Furthermore, the 3.03-μm diameter particles are collected at 99 percent, and thus, these particles contribute an additional 1.4 percent to the measured collection of particles on the impaction plate.

Therefore, the collection efficiency of 2.1-μm diameter particles is actually only 1.8 percent instead of the 11.3 percent originally thought. The correct calibration curve is, shown in Figure 3), the correction becomes quite large, giving the indication of an efficiency curve with a large "tail" at the lower efficiencies when actually only a small tail exists in the "true" collection efficiency curve.

The efficiency curves determined with this technique are shown in Figure 4 for both the 2.5 and 10-μm cut point impactors as dashed lines. The corresponding theoretical efficiency curves[5] are shown as solid lines. Inspection of these curves shows close agreement between theory and experimental results. If the sharpness of cut is defined by the geometric standard deviation (σ_g), where the σ_g is equal to the square root of the ratio of the particle diameter corresponding to the 84.1 percent collection efficiency to the diameter at an efficiency of 15.9 percent, it is found to be 1.02 and 1.11 for the 2.5 and 10-μm curves, respectively.

As shown in Figure 3, the multiplet correction influences the shape of the collection efficiency curve more than the 50 percent cutpoint when the impactor has sharp classification characteristics. However, for impactors which do not have sharp classification characteristics, the multiplet correction

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can also effect the 50 percent cutpoint. For example, if the sharpness of cut of an impactor efficiency curve is characterized by a geometric standard deviation of 1.6, the 50 percent cutpoint would be decreased by about 5 percent if there were 9 percent doublets. Although this is a small shift in the efficiency curve, it is a correction that should be considered when calibrating impactors with monodisperse aerosols.

Interstage Losses

Interstage losses are defined as the fraction of particles of a particular size that are collected at any point other than the impaction plate or afterfilter. Since the losses are a weak function of particle size, the losses at only a few particle sizes were measured.

Afterfilter Deposit Uniformity

One of the design criteria for the impactor was for the particles to be uniformly deposited on the afterfilter. Uniformity was checked by dividing the filter into five annular areas and measuring the dye concentration per unit area from the particles washed from the filter segments.

Uniformity of the particle deposit on the afterfilter was achieved by allowing adequate distance between the second stage impaction plate and the filter. It was found that 2 cm was sufficient for uniformity to be achieved with the 2.5-μm cut impactor with 4 percent variation which was the approximate uncertainty in the uniformity measurement technique. For the 10-μm cut impactor, the deposit on the afterfilter was not uniform with the 2-cm length. Therefore, the final design, as shown in Figure 2, has a tube length of 10.5 cm. This length is more than sufficient to achieve a uniform particle deposit on the afterfilter in the 2.5-μm version and provides adequate uniformity for the 10-μm version.

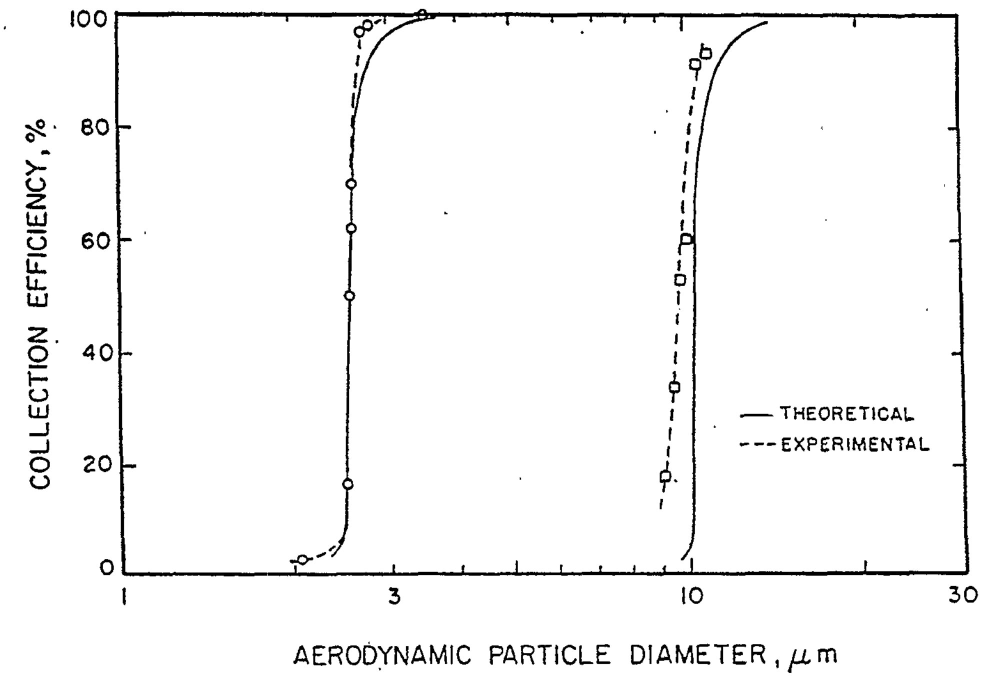

Figure 4. Theoretically and experimentally determined collection efficiency curves for the 2.5 and 10 μm cutpoint sampler.

The fraction of particles lost is measured in exactly the same manner as the fraction collected on the impaction plate; i.e., the internal surfaces are washed with known amounts of 0.001 N sodium hydroxide. The resulting dye concentration measured with a fluorometer provides good information on the fraction of particles lost.

Losses for three particle sizes of 2.0, 2.5 and 2.7 μm diameter were found to be 0.2 percent, 0.2 percent and 0.1 percent, respectively, for the 2.5-μm cut size impactor. Since losses near the cut size are expected to be the largest, the losses for this impactor are negligible.

Losses in the 10-μm cut size impactor were measured at 9.3 μm diameter and found to be 0.07 percent. Since losses were so small at this size, no other loss tests were performed and losses in the 10-μm cut impactor are considered negligible as well.

Conclusions

An impactor operating at a flow rate of 4 L/m for indoor air sampling has been developed. Two versions were designed; one with a cut size of 2.5 μm and one with a cut size at 10 μm. The particles larger than the cut size were removed from the air stream and not used, while the smaller particles were collected on an afterfilter. The afterfilters are subsequently analyzed for mass gain and/or chemical composition. Two stages of the same cut size were used in a cascade fashion for both versions of the impactor to minimize parti-

cle bounce problems. The use of oiled porous impaction plates further reduced particle bounce. Calibration of both versions of the impactor showed sharp cutoff characteristics, low interstage losses and a uniform deposit on the afterfilter. To increase the rate of particle collection, two new impactors are being developed with increased flow rates (10 L/m) while keeping the cut sizes the same (2.5 μm and 10 μm diameter).

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THE EFFECTS OF HVAC SYSTEM DESIGN AND OPERATION ON RADON ENTRY INTO SCHOOL BUILDINGS

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ABSTRACT

Heating, ventilating, and air conditioning (HVAC) systems in schools vary considerably and tend to have a greater impact on pressure differentials -- and consequently radon levels -- than do heating and air-conditioning systems in houses. If the HVAC system induces a negative pressure relative to the subslab area, radon can be pulled into the building. If the HVAC system pressurizes the building, it can prevent radon entry as long as the fan is running. However, school HVAC systems are normally set back or turned off on evenings and weekends and, even if the HVAC system pressurizes the school during operation, indoor radon levels may build up during setback periods.

Many of the historical methods utilized to deliver ventilation air (outdoor air) over the past 40 years are summarized. In addition, for each type of system presented, the possible impact the ventilation system might be expected to have (positive or negative) on the pressure of the building envelope (and subsequent radon levels in the building) is discussed.

This paper has been reviewed in accordance with the U.S. Environmental Protection Agency's peer and administrative review policies and approved for presentation and publication.

INTRODUCTION

THE EFFECTS OF VENTILATION ON BUILDING PRESSURE DIFFERENTIALS

The primary mode of radon entry into a school is normally from soil gas that is drawn in by pressure differentials between the soil surrounding the substructure and the building interior. If the building interior is at a lower pressure than the soil surrounding the substructure, radon can be pulled in through cracks and openings that are in contact with the soil. The amount of radon in a given classroom will depend on the level of radon in the underlying material, the ease with which the radon moves as a component of the soil gas through the soil, the magnitude and direction of the pressure differentials, the number and size of the radon entry routes, and dilution and mixing of the room air.

Pressure differentials that contribute to radon entry can result from operation of a heating, ventilating, and air conditioning (HVAC) system under conditions that cause negative pressures (in the building relative to the subslab area), indoor/outdoor temperature differences (including the "stack effect"), use of appliances or other mechanical devices that depressurize the building, and wind.

HVAC systems in schools and other large buildings vary considerably and tend to have a greater impact on radon levels than do heating and air-conditioning systems in houses(1). The design, installation, and operation of the ventilation equipment will cause the building envelope to be at a positive, negative, or neutral pressure with respect to the outdoors, depending on the system design, how it was initially installed and balanced, and how it has been historically maintained and operated. Sometimes schools and similar buildings were not designed with adequate ventilation, and in other instances, ventilation systems are not operated properly due to factors such as increased energy costs or uncomfortable conditions caused by a design or maintenance problem. Such circumstances may enhance radon entry into the building.

If the HVAC system induces a negative pressure in the building relative to the subslab area, radon can be pulled into the building through floor and wall cracks or other openings in contact with the soil. (Even if the HVAC system does not contribute to pressure differentials in the building, the natural stack effect in a leaky building can cause the building to be under a negative pressure so that radon-containing soil gas is pulled into the school.)

If the HVAC system pressurizes the building -- which is a common design in many systems -- it can prevent radon entry as long as the fan is running. However, school HVAC systems are normally set back or turned off during evenings and weekends, and even if the HVAC system pressurizes the school during operation, indoor radon levels may build up during setback periods. Once the HVAC system is turned back on, it may take several hours for radon levels to be reduced. Consequently, how the ventilation air (outdoor air) is supplied to the building (i.e., whether the ventilation system is pressurizing or depressurizing the building) can be expected to drastically affect the radon levels in a building when the ventilation system is operating. Even when a building is operated overall slightly positive with respect to the outdoors, localized

negative pressures may exist. If openings to the earth are present where these localized negative pressures exist, soil gases will be drawn in.

BUILDING VENTILATION HISTORY

Buildings designed for human occupancy (in particular, public buildings such as schools) have historically been required to be designed to provide ventilation air (outdoor air) to the occupants. This outdoor air in early designs was provided by non-powered design features such as operable windows and gravity ventilators (which allow air movement created by wind and temperature differences), more recently by powered ventilating equipment, or some combination of both. In many areas of the country where air conditioning has not been utilized, it has been popular to provide a base level of ventilation by mechanical systems and to allow supplemental ventilation to occur through the use of large operable windows as weather conditions allow.

Historically, the introduction of fresh outdoor air into buildings has been relied upon to dilute the contaminants which are generated by the occupants within the building and to provide free (economizer) cooling when weather conditions permit. The American Society of Heating, Refrigerating and Air Conditioning Engineers (ASHRAE) standards documents have provided, and continue to provide, engineering professionals with guidelines to the suggested minimum quantities of outdoor air which should be provided to the occupants (2). Building Codes (laws) also govern the amounts of outdoor air to be provided; often these codes refer to the ASHRAE guidelines. The most up-to-date ASHRAE ventilation standard, ASHRAE 62-1989, prescribes that 15 cfm (1 cfm = 0.47 L/s) of outdoor air per student be supplied to a classroom for acceptable indoor air quality (2).

MULTIPLE ZONING OF VENTILATION AIR

In large buildings with multiple exhaust fans, supply air systems, and system balancing dampers, some sections of the building will frequently be designed to be operated negative and other sections positive with respect to adjoining areas in order to minimize the spread of an internally generated irritant or odor. Thus, with regard to radon entry from the soil, the expected and measured overall pressure balance of the total building envelope and the expected and measured pressure relationship in individual areas of the building must be considered.

Typical areas which might be expected to be designed and operated negative with respect to adjoining areas include any area where identifiable sources of pollutants may be generated; e.g., toilets, locker rooms, shops, print rooms, art areas, laboratories, kitchens, gymnasiums, hallways, lounges, and janitor's closets. Areas which might be expected to be designed and operated positive with respect to adjoining areas include classrooms, computer rooms, and libraries. Thus, it is important to know the expected and measured pressure relationship of individual zones within the envelope as well as the overall building envelope.

In addition to affecting the pressure relationships, the ventilation air (outdoor air) will also be available to dilute radon gas once it has entered the building. The dilution effect of outdoor air (ventilation air) is primarily

a control strategy for other pollutants (bioeffluents) generated primarily by the occupants; however, dilution alone is seldom adequate to reduce elevated levels of radon without the proper pressure relationship.

The following section presents an overview of the various types of ventilation systems which might be found in school buildings and the potential (positive or negative) impact each type of system would be expected to have with regard to radon entry (i.e., the expected overall impact on the pressure relationship between the building envelope and the soil).

TYPES OF HVAC SYSTEMS

Many of the HVAC systems discussed below have the option of being designed to supply a fixed or variable amount of outdoor air. In addition, the total supply air moved by an air handling system (i.e., the combination of outdoor and recirculated air) may be fixed (constant volume) or modulated (variable volume). A variable air volume (VAV) system is typically designed to deliver more total supply air as additional cooling is called for. With a VAV system, the amount of outdoor air delivered may also be designed to be fixed or modulated.

EXHAUST-ONLY SYSTEMS

One of the basic systems is an exhaust-only system; i.e., the system consists of exhaust fans (often installed in hallways, bathrooms, and locker areas). Building leakage or the opening of windows is typically the source of outdoor makeup air. Even more basic systems include gravity ventilators (non-powered exhaust shafts dependent upon the building stack effect, and operable windows). Such systems that do not supply tempered makeup air typically lead to stuffy conditions in the winter time, when occupants are hesitant to open windows due to cold drafts.

Exhaust-only systems would be expected to cause the overall pressure within the building to be negative with respect to the outdoors, thus increasing the flow of soil gas into the building envelope. Depending on the degree of building depressurization, and the location and size of the envelope leakage, the radon levels in a building should increase during operation of an exhaust-only system.

RADIANT HEAT SYSTEMS

Radiant heat systems in schools tend to be of three types: hot water or steam radiators, baseboard heaters, or warm water radiant heat within the slab. Schools heated with radiant systems should have a ventilation system to achieve the fresh air requirements recommended by ASHRAE; however, many of these schools provide no ventilation other than natural infiltration or sometimes a few operable windows. In other schools, there are exhaust ventilators on the roof. These can be passive, allowing some ventilation through the stack effect, or they can be powered. Powered roof ventilators (PRVs) can cause significant building depressurization, particularly if a fresh air supply is not provided. This can cause considerable radon entry into the building while such exhaust systems are operating.

UNIT VENTILATORS

The use of unit ventilators in schools has been and continues to be very popular. They are available in a number of different arrangements: horizontal, vertical, draw-through, and blow-through; and are made by a wide variety of manufacturers.

In a typical unit ventilator system, by design, there is a connection to the outdoors, providing makeup air for ventilation and free cooling. In most unit ventilator configurations, the outdoor air mixes with return air from the classroom in the plenum portion of the unit ventilator and is supplied to the space through the top.

The advantages of this type of system are often economics and architectural flexibility: generally no ductwork is required. Some of the disadvantages of this system are the noise levels generated by the unit ventilators and the numerous wall penetrations that at some points downgrade the architects' elevation aesthetics. Also, a serious concern is the draftiness of these types of units especially in northern climates. Drafts are of concern because, with 20 to 25 students in a typical modern classroom, coupled with well insulated walls and ceilings and 1.5-2 W/ft^2 (335-446 kW/m^2) of lighting, the internal heat gains often outweigh any envelope losses of the classroom. This can require fresh air to be introduced for cooling during major portions of the school year.

Unit ventilator systems would be expected to cause the overall pressure within the building to be positive with respect to the outdoors, thus reducing the flow of soil gas into the building envelope when the unit is operating and outdoor air is being drawn into the unit. Whether or not the space served by the unit ventilator is actually pressurized with respect to the soil will depend on the degree of overall building pressurization or depressurization. If other areas of the building have exhaust-only systems which exhaust more air than is made up by the unit ventilators, then soil gases will still be drawn into the building in areas where the net pressure is negative. The radon levels in a building should decrease when a unit ventilator system is operating properly, if adequate overall makeup air is provided.

TERMINAL AIR BLENDERS

Terminal air blenders have also been used. Initially, this type of system was a good alternative to unit ventilators. There are a number of ways terminal air blenders have been used. They were installed to help combat the energy crunch, while still delivering outdoor air for cooling and ventilation. With these systems in the classroom, even in northern climates, over 90 percent of the time approximately 5 cfm (0.00236 ft^3/s) of outdoor air per student would typically be introduced. Because of the high internal heat gains in the classroom, the intent was to thermostatically control the outdoor air quantity, bringing in the appropriate amount of outdoor air required to satisfy the internally generated heat load. These systems are generally connected to an air duct system to distribute the ventilation air evenly, reducing drafts, and are less noisy than unit ventilators. A consistent outdoor air supply is not provided; however, typically, 90 percent of the time more than 5 cfm per student

outdoor air is provided with a thermostatically controlled terminal air blender ventilation system that is functioning properly.

Terminal air blender systems would be expected to cause the overall pressure within the building to be positive with respect to the outdoors, thus reducing the flow of soil gas into the building envelope when the unit is operating and outdoor air is being drawn into the unit and distributed to the occupants. Whether or not the space served by the terminal air blender is actually pressurized with respect to the soil will depend on the degree of overall building pressurization or depressurization. That is, if other areas of the building have exhaust-only systems which exhaust more air than is made up by the terminal air blender, then soil gases will still be drawn into the building in areas where the resultant pressure is negative. The radon levels in a building should decrease when a terminal air blender system is operating properly, if adequate overall makeup air is provided.

UNITARY HEAT PUMPS OR FAN-COIL UNITS

Heat pump units have been utilized to a limited degree in schools. They appear similar to a fan-coil unit and may or may not have outdoor air ducted to the unit. Fan-coil units consist of a fan and heating and/or cooling coils and may or may not have outdoor air ducted to the unit. (They may just recirculate air.)

Unitary heat pumps or fan-coil units would be expected to cause no overall pressure change within the building even when outdoor air has been ducted to the unit unless additional dampers and controls have been added to convert it to function as a unit ventilator. If outdoor air has been provided and the unit converted to a unit ventilator, then the unit would be expected to cause a positive pressure inside the building with respect to the outdoors, thus reducing the flow of soil gas into the building envelope when the unit is operating and outdoor air is being drawn into the unit and distributed to the occupants. Whether or not the space served by the unit is actually pressurized with respect to the soil will depend on the degree of overall building pressurization or depressurization. That is, if other areas of the building have exhaust-only systems which exhaust more air than is made up by the heat pump or fan-coil units, then soil gases will still be drawn into the building in areas where the net pressure is negative. The radon levels in a building should decrease only when a fan-coil or heat pump unit has been equipped with outdoor air, and converted to a unit ventilator (if adequate overall makeup air is provided). If the units are not supplied with outdoor air then the only impact should be from the normal natural stack effect of a leaky building.

HEAT RECOVERY VENTILATORS (HRV)

In general, HRVs are either ducted systems with supply and return ducts servicing different parts of the building or room, or wall-mounted units, similar to wall-mounted air-conditioning units. In both types of units, fresh air is brought in through a heat recovery device, then distributed, or passed through a preheat coil and then out to the system's zones. The exhausts from these zones pass through a separate section of the heat recovery device, and then discharge, far enough from the fresh air intake to minimize re-entrainment. One

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disadvantage of these types of systems is that condensation on the surface of the heat exchanger may frost up and block the heat exchanger when outdoor temperature drops below 20°F [°C=5/9(°F-32)]. To avoid that problem, bypass sections and defrost controls are often available with units as standard features. By temporarily bypassing the outdoor air and, thus, raising the exchange surface temperature to above the dewpoint, frost on the exchanger surface is avoided.

If balanced correctly, HRV systems would be expected to cause the overall pressure within the building to be neutral or very slightly positive with respect to the outdoors, thus reducing the flow of soil gas into the building envelope when the unit is operating and outdoor air is being drawn into the unit and distributed to the occupants. Whether or not the space served by the device is actually pressurized with respect to the soil will depend on the degree of overall building pressurization or depressurization. That is, if other areas of the building have exhaust-only systems which exhaust more air than is made up by the HRVs, then soil gases will be drawn into the building in areas where the resultant pressure is negative. The radon levels in a building should decrease when a HRV system is operating properly, if adequate overall makeup air is provided. One exception is exhaust-only heat recovery devices which would be expected to raise radon levels similar to exhaust-only ventilation systems discussed earlier.

CENTRAL STATION AIR HANDLERS

There are many types of central station systems, many with features similar to those discussed above. The common features of all central units include: an air handling unit, supply fan and/or return fan, and associated tempering coils, air filters, dampers and controls, distribution ductwork, exhaust (or relief), mixing box, and outdoor air intake. In the past, constant volume systems, which consisted of central station air handling systems that generally had fixed minimum outdoor air dampers, were used in schools. Typically the outdoor air would be controlled by a two-position damper closed and opened to whatever percentages were predetermined, to be mixed with return air, passed through the supply fan, and introduced to the occupied space.

If designed correctly, central station air handler systems would be expected to cause the overall pressure within the building to be slightly positive with respect to the outdoors, thus reducing the flow of soil gas into the building envelope when the unit is operating and outdoor air is being drawn into the unit and distributed to the occupants. However, in a building with multiple zones, some spaces served by the central system may be adjusted to be positive with respect to the soil, and other areas may be negative. Many areas of the building may have exhaust fans which exhaust more air than is made up by the central system by design. Thus, even if the overall pressure relationship for the building is positive, soil gases will still be drawn into the building in areas where the local resultant pressure is negative. The radon levels in a building should decrease when a central station system is balanced to be slightly positive and operated properly, if adequate overall makeup air is provided. The following sections present a few of these central station air handler systems in detail.

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Conventional Constant Volume (CV)

Central stations, predominantly with constant volume air systems with fixed minimum outdoor air entry and reheat coils, have been utilized for many years in a limited number of schools. They are somewhat energy efficient and are easily balanced.

Variable Air Volume (VAV)

With the energy crunch, several manufacturers of air handlers introduced VAV control. The most immediate savings are fan energy and elimination of reheat coils (if individual room VAV diffusers are used); however, outdoor air control is difficult. In many VAV systems, there is no way to control outdoor air to bring the room above bare minimum fresh-air quantities. Central station VAV systems, with static pressure devices in the outdoor air stream and addition of reheat coils, were an answer to outdoor air control. With static pressure control of the outdoor air stream, it is possible to maintain an overall positive pressure within the building under various operating conditions.

VAV with Economizer

As just noted, the VAV helps cut down on fan energy when not dealing with peak cooling loads. If only minimum air movement is needed, the reduced air flow will save energy dollars. Whenever outdoor air is critical (e.g., if all areas must be kept under positive pressure to keep radon out), shutting off the VAV distribution boxes in individual spaces is a concern. One disadvantage of most VAV systems is that they have no sensing in the outdoor air stream that would guarantee the correct amount of outdoor air during part-load operation. One way to avoid this situation is to use an outdoor air flow sensor. With this type of metering system, a drop in velocity in the outdoor air stream will control the air dampers, bringing in more air from the outdoors. (This would be typically called an outdoor air reset.)

VAV with Outdoor Air Control and Heat Recovery

This type of package combines efficient operation with temperature control. Most importantly, the ability to deliver outdoor air capacity is greatly increased, and the facility is not penalized in terms of energy costs, nearly as much as without the heat recovery feature.

Central station heat exchangers are currently being considered in many schools being designed for northern climates. In this type of design, a central air handling system incorporates a heat exchanger. Some reheat may be required in such system.

HVAC SYSTEMS AND RADON MITIGATION

A potential mitigation approach for schools is adjustment of the air-handling system to maintain a positive pressure in the school relative to the subslab area, discouraging the inflow of radon. This technique, referred to as pressurization, has been shown to be an effective temporary means of reducing radon levels in some schools, depending on the design of the HVAC system. If

pressurization through the HVAC system is under consideration as a long-term radon mitigation solution in a given school, proper operation and maintenance of the system are critical. Responses to changes in environmental conditions and any additional maintenance costs and energy penalties associated with the changes in operation of the HVAC system must also be carefully considered(3).

Important factors that need to be considered when utilizing the HVAC system for radon control include: (1) How much outdoor air was the system originally designed to supply under what design conditions? (2) How leaky is the shell of the building and can pressurization be utilized? (3) Is the system currently operating as designed, or has it been modified purposely or through neglect? and (4) How will moisture effect the building shell? Once the limitations of the HVAC system and building shell are determined, decisions can be made on the best or most reasonable course of action which can be taken. Some approaches to radon control through the HVAC system that have been used temporarily and permanently are generalized below.

EXHAUST-ONLY VENTILATION AND RADIATION HEAT SYSTEMS

For schools with either exhaust-only ventilation systems or radiant heat, positive pressurization will probably require major modifications if the HVAC system is considered as part of the mitigation strategy.

UNIT VENTILATORS AND EXHAUST-ONLY SYSTEMS

Radon mitigation strategies in schools with unit ventilators might include (1) opening the fresh-air vents (if they have been closed) to improve ventilation and running the unit ventilator fans continuously (or prior to occupancy) to pressurize the room; (2) replacing an exhaust-only ventilation system with a system that operates under a slight positive pressure; or (3) installation of a subslab depressurization (SSD) system that could overpower all negative pressures in the building. If the current HVAC system is providing adequate ventilation to the building or if options (1) and (2) are not feasible, option (3), installation of a SSD system, would be the most practical near-term strategy if there is good subslab communication.

CENTRAL AIR HANDLING SYSTEMS

Although most central HVAC systems are commonly designed to operate at positive or neutral pressures, pressure measurements in schools have indicated that such systems can cause significant negative pressures in the building. HVAC system modifications (such as reducing the amount of fresh-air intake), lack of maintenance (such as dirty filters), unrepaired damage, or other factors can result in substantial negative pressures in some rooms, thus increasing soil gas entry. In addition, operation of localized exhaust fans can cause significant negative pressures in areas of operation.

If positive pressures are not being achieved in a central single-fan system, the system should be checked to ensure that the fresh-air intake meets design specifications and that the intake has not been closed or restricted. Increasing the fresh-air intake if it has been restricted, and operating the fan for a sufficient time prior to occupancy and continuously while the school is occupied

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will help to reduce radon levels that have built up during setback periods and will maintain low radon levels during occupied hours by preventing radon entry by maintaining a positive pressure and by providing fresh (dilution) air.

In a central dual-fan system, the return-air fan can be set back or restricted so that all of the rooms are under a positive pressure. The fresh-air intake to the supply fan can also be increased up to the design limit of the system, if it has been reduced. If radon control through HVAC system operation is under consideration as a permanent mitigation strategy, proper system operation and maintenance are critical.

Many schools with highly elevated radon levels have installed SSD systems in order to control radon levels even when the HVAC system is not operating.

ACKNOWLEDGEMENTS

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ACHIEVING TOBACCO SMOKE-FREE ENVIRONMENTS, THE PROBLEM, AND SOME SOLUTIONS

By: David W. Bearg, P.E.
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ABSTRACT

Tobacco smoke has long been recognized as a serious irritant and potential health hazard to non-smokers in the indoor environment. Various solutions to this problem have been implemented by many managers with both success and failure. This paper presents cases in different buildings where tobacco smoke was identified as an environmental irritant and illustrates different control strategies that did and did not eliminate the problem. Methods and results are detailed.

INTRODUCTION

There has been increased attention focused on achieving environments that are free from the air contaminants associated with environmental tobacco smoke (ETS). This has occurred in response to the 1986 Surgeon General's Report entitled, "The Health Consequences of Involuntary Smoking" and the National Academy of Sciences Report entitled, "Environmental Tobacco Smoke Measuring Exposures and Assessing Health Effects." These reports have both concluded that ambient tobacco smoke can and does cause lung cancer and other serious health problems for nonvoluntary smokers. This issue is not a new one and the authors have been involved with this aspect of indoor air quality investigations for a number of years, focusing on objectively determining the impact of smoking activities on nearby areas which are intended to be smoke-free. This paper presents a summary of the information we have gained from these experiences.

The control of any air contaminant, such as environmental tobacco smoke, can typically be treated by one of the five basic options of control: source removal, source modification, air cleaning, dilution ventilation or exhaust ventilation. This paper discusses the application of each of these approaches for achieving smoke-free environments in office-type buildings.

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SOURCE REMOVAL

From an engineering standpoint, the most efficient technique for achieving a tobacco-smoke-free environment is the elimination of smoking activity from the given environment. That is the trivial solution. This presentation therefore will focus on the applications of the other options either singly or in combination.

SOURCE MODIFICATION

The second option, source modification, refers in this discussion to the relocation of smokers from many locations to only one or a few specific locations to achieve a reduction in the number of areas directly impacted by ETS. This approach, by concentrating the number of smokers in fewer locations, may change the magnitude of exposures and rearrange the distribution of ETS but will not achieve a smoke-free environment if the area or areas where smoking is permitted is connected to an HVAC system which recirculates the return air to other locations in the building which are intended to be smoke-free.

Our testing efforts have demonstrated that the simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate, exposure of nonsmokers to environmental tobacco smoke. This is also a major conclusion of the Surgeon General's report (1986).

Examples from our testing experience point out the problems with this approach. In one hospital cafeteria, about one-third of the area was designated "smoking" while the remaining two-thirds was designated as "nonsmoking." The impact of ETS was determined by measuring the concentration of respirable particulate matter (RSP), one component of ETS which provides a useful analytical indicator. These RSP concentrations were measured by a portable mass respirable monitor (using a piezoelectric quartz crystal for determining the relative RSP concentrations). In this situation, as with other cafeterias, the distribution of RSP was largely influenced by the airflow patterns created by the presence of exhaust fans in the food preparation area. The resulting distribution in this example consisted of elevated RSP concentrations over most of the cafeteria, especially in the nonsmoking area situated between the smoking area and the food service area. In another cafeteria arrangement, the nonsmoking area was located entirely between the food-serving line and the smoking permitted section. This geometry meant that the entire nonsmoking section, not just a portion of it, was impacted by the adjacent smoking activity. These examples point out the great difficulty in providing smoke-free environments in cafeterias because of the influence of the exhaust systems in the food preparation areas. For instance, if the smoking area were closer to the kitchen, then the nonsmokers on the food-serving line would be impacted by the nearby smoking. In both

these situations, because it was the intent of both hospital administrations to provide their employees and visitors with a smoke-free environment, smoking was entirely banned from these cafeterias. A major redesign of the ventilation system was not a viable option in these cases.

In two other cafeteria examples, the influence of a recirculating cooling system was demonstrated. In one system, there were four fan/coil units above the ceiling that only served the cafeteria. The air intakes for these four parallel systems were located in the ceiling near the food serving lines of the cafeteria with the return being to the cafeteria via three or four ceiling diffusers that are distributed the length of the room. The measurements of RSP in this situation demonstated that by keeping the room well mixed, the area concentrations rose uniformly throughout the room. The only difference between the nonsmoking and smoking areas was that the smoking area contained several higher peak RSP concentrations in the immediate vicinity of smokers. This HVAC system did not provide any observable reduction in the concentrations of RSP. The second example differed in that the nonsmoking area was more physically separated from the smoking area by enclosing walls. However, since there was not a separate ventilation system as well, the measured RSP concentrations in the nonsmoking area were again similar to those obtained in the smoking area.

AIR CLEANING

The possibility does exist, however, that the HVAC could remove the ETS air contaminants from the return air stream before recirculating all or a portion of this air back to occupied spaces. This approach leads to the third option, air cleaning, which refers to the use of equipment for removing the air contaminants from cigarette smoke as part of either the HVAC system or as an isolated installation near the site of the smoking activity. ETS is characterized not only by respirable particulates but also benzene, acrolein, N-nitrosamine, pyrene, nicotine decomposition products, and carbon monoxide. Air-cleaning equipment includes HEPA filters or electrostatic precipitators, which can remove the respirable particulate from the airstream, but have not demonstrated the ability to remove the volatile components, nor should they be expected to. Collection of volatile components would require the use of a granulated filter media capable of adsorption such as activated carbon or some type of catalytic system.

Research has shown that the semi-volatile components of ETS, which may initially be collected by the air-cleaning device (due to their adsorption onto the particulate matter), may be released into the airstream. The presence of these semivolatile components of ETS may then still be a source of potentially irritating odors to nonsmokers.

In one example evaluated, isolated ceiling-mounted electrostatic air cleaners were installed in hospital "day rooms." The use of this equipment did not yield a measurable improvement in reducing the concentrations of RSP as compared to when the equipment was not in use. Even if there had been a measurable reduction in RSP concentrations, this would not be sufficient to characterize the space as smoke-free, because there would be no reduction in the volatile components of ETS which were still being released into and recirculated around the room. Electrostatic precipitators (ESPs) require frequent cleaning to maintain their effectiveness and minimize the release of odors from previously collected material. The ESP installations observed by the authors have typically not been receiving cleaning on a regular or frequent basis. Installing the air-cleaning device in the HVAC system as opposed to merely locating it in ceiling above a smoking area may increase or decrease the likelihood of frequent periodic cleaning depending on who is responsible for this maintenance. In-duct installations possess the same problems. Articles have been written which attempt to assess the effectiveness of this approach based on chamber studies (Allen H. Frey, 1985). In the opinion of the authors, the overall effectiveness of this approach in controlling both RSP and odors has yet to be proven.

DILUTION VENTILATION

One difference of in-duct installations of air-cleaning devices, as compared with site specific installations, is that they may further reduce ETS concentrations by diluting the air contaminants into a larger volume of air. The down side of this is that a larger portion of the building may be impacted. This brings us to a discussion of the next basic option for the control of air contaminants -- dilution ventilation. Dilution ventilation refers to dilution of contaminated air with uncontaminated air in a general area, room, or building for the purpose of health hazard or nuisance control (American Conference of Governmental Industrial Hygienists, 1984). Since ETS has now been identified as a cause of cancer, the question can be raised as to whether any amount of dilution would achieve a zero threshold of exposure. As stated in the Surgeon General's report, "The first response to the identification of a carcinogen in the work environment should be to eliminate that exposure. It is only when elimination of the exposure is not possible that we should explore establishing acceptable levels for the worksite." One approach for dilution ventilation would be to determine what ventilation rates would be required so that visitors to an area would not object to the odor of tobacco smoke. Work of this nature was performed by Cain and Leaderer (1982) who reported that ventilation rates up to 30 cfm per occupant were not sufficient to achieve a "75-80% acceptance of the odor by visitors" which was their criterion for acceptability. They concluded that "a ventilation rate as high as 100 cfm per smoking occupant might be necessary" to meet their criterion of acceptability in situations where smoking occurs more or less continuously.

This article also reported that "surfaces in an enclosed room, seem to be important sinks for tobacco smoke indoors" and, "that absorbed particles may carry condensed volatiles which could evaporate over time, thereby imparting a lingering odor". To prevent this contamination of surfaces in a smoking area, it is recommended that the preferred method of control be the application of local exhaust ventilation.

EXHAUST VENTILATION

This approach is an embodiment of one of the basic principles of industrial hygiene, the collection of air contaminants as close to the source as possible. This approach minimizes the air volumes required and maximizes the collection efficiency. This approach also requires that quantities of make-up air equal in volume to that being exhausted be supplied to the vicinity of the exhausted area. Aside from the complete elimination of smoking activities, this approach has the greatest potential for achieving tobacco smoke-free environments. To achieve the desired goals there are certain basic rules that need to be followed. The first requirement is the designation of a specific location as a smoking lounge. This location needs to be a physically enclosed space that is separate from other areas. This includes isolation from the rest of the recirculating air-handling system to prevent the transport of ETS air contaminants to other locations in the building. This can be achieved by blocking the return-air ductwork located in this area and replacing it with exhaust registers that transport the air contaminants directly to the outside for discharge. The exhaust fan for this system should also be located outside or as close to the outside as possible so that the ductwork transporting the air contaminants is under negative pressure in the building. This is important because if the ductwork is not leaktight and under positive pressure, there can be leakage of air contaminants back in the return plenum. It is also important to locate this point of discharge away from any air intakes to minimize the possibility of reentrainment. The location of both the exhaust register and the makeup supply air is critical. It is crucial that they be located so as to minimize any short circuiting of the supply air directly to the exhaust. One example of short circuiting was observed at a school where smoking was permitted in one of the two teacher's lounges. In this situation, the exhaust register was located in the ceiling near a corner of the room and the make-up air came through a grille in the door to the room. Unfortunately for the effectiveness of the system, the door to the room was located in one of the walls nearest the exhaust register thereby eliminating much of the potential benefit of this exhaust ventilation. A more effective geometry for the collection of ETS air contaminants in this situation would have been to have the exhaust register still in the ceiling but near the wall furthest from the location of the supply air. In a situation where there is a ducted supply system, collection efficiencies would be maximized and the greatest likelihood of achieving a smoke-free environment would occur

if the make up air were supplied near floor level and at several locations around the room. This strategy has yielded successful results for one of our clients. The exhaust register should be in the ceiling, either in the center of the room or as close to the maximum concentration of smokers as possible.

Another advantage of exhaust ventilation, as compared with dilution ventilation, is that with the proper placement of exhaust registers and supply grilles, it is possible to achieve plug-flow through the space. This situation will achieve the maximum benefit of air contaminant removal for given quantities of airflow. This is particularly important both for energy conservation and in situations where the "smoking lounge" is a multiple-use space and will be occupied by non smokers as well as smokers.

CONCLUSIONS

The easiest approach for achieving a smoke-free environment for workers would be to eliminate smoking entirely from the building where they work. Exposures to air contaminants from ETS can be reduced by means of air-cleaning techniques or dilution ventilation, however, to be effective in achieving a smoke-free environment, there needs to be a local exhaust ventilation system which both isolates a smoking area from all other areas and discharges the ETS air contaminants outside and away from all air intakes.

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DELIVERING "DESIGNED" CFM OF OUTSIDE AIR TO THE OCCUPANTS, PROBLEMS AND SOLUTIONS

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ABSTRACT

The American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) recently proposed an increase in the minimum amount of outside air that should be designed for and delivered to occupants in indoor office-type environments. This paper will present the assessments of several buildings in which ventilation was "designed" to be adequate but, when field tested years later in response to complaints of indoor air quality problems, was found to be incapable of delivering the design quantities of ventilation air. These situations were often caused by measured short circuiting in the heating, ventilating and air conditioning system, improper installation of HVAC equipment, or poor preventive maintenance. The results of corrective measures are detailed.

INTRODUCTION

In the process of conducting indoor air quality investigations in office-type buildings, the authors have identified several important areas that need to be evaluated. These include, but are not limited to, the history of the occupant complaints, the distribution of outside air to the occupants, and the sources of potential irritants located both inside and outside the building. The overall techniques utilized in conducting these investigations are described elsewhere (Bearg and Turner 1985, 1987). This paper focuses on the evaluation of the amount of outside air being distributed to the occupants.

In determining the amount of outside air made available to the occupants, we have identified three important questions:

1. What was the space originally designed to be? (What was the original intent of the design, based on drawings, specifications, and design quantities or does anybody know?)

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2. What is it currently being used for?
(Based on inspection and defensible measurements, what is really happening there?)

3. What do we want to be happening?
(Based on current and planned space usage and the current and evolving guidelines concerning the quantitites of outside air that are considered desirable to deliver to occupants, what should be happening?)

ASSESSMENT METHODOLOGIES

Original Design

The determination of the original intent of a design (i.e., what was supposed to be happening in this space) may be as simple as finding the original design drawings and specifications and extracting information. For older buildings, this can be a tedious chore and success may be determined by luck, not by skill. The task is often easiest when the building is owner-occupied, and the original owner has been there since the building was constructed.

Historic and Current Situation

The determination of what is currently happening in this space, and possibly in the recent past, is a matter of meticulous inspection, testing, and measurement. By inspection and communication with the owner, one can often determine the building's actual occupancy, space utilization, and evidence of HVAC preventive maintenance. Historical conditions can be inferred from the situation as found. The actual amounts of outside air being delivered to a space, however, can be evaluated only by testing and measurement. This testing must, at a minimum, include the inspection and testing of all controls that affect the delivery of ventilation and outside air to the occupants and the actual measurement of the quantities of outside air and/or total air in question. Equipment and techniques that the authors have found useful in producing defensible results include the use of velocity-measuring devices such as pitot tubes and airflow hoods and anemometers, temperature and humidity probes for determining enthalpy balances at HVAC air-handling units that supply outside air, and static pressure sensors for determining pressure relationships within air handling units and building envelopes. All equipment utilized must be calibrated in order to develop accurate, defensible results. The approach that the authors have found indispensible in assessing the amount of outside air actually delivered to occupants is the combined use of tracer gas measurements, enthalpy balance testing, and velocity measurements. The combination of these techniques will allow both measurement of designed outside air flows and supplemental infiltration/exfiltration to be addressed if conducted under appropriate weather conditions.

The tracer techniques utilized have included a modified version of the ASTM methodologies and methodologies currently being developed by National Bureau of Science (NBS) researchers (ASTM 1980; Persily and Grot 1985). Sulfur hexafluoride (SF6) is the tracer most often utlized by the authors. It has been chosen because of its detectability at very low levels with portable gas chromatographic equipment, its inert nature, and its limited normal usage in occupied buildings. Another technique that has been useful as a survey tool in assessing areas of potentially low outside air supply is the utilization of carbon dioxide (CO2) measurements, using a real-time calibrated CO2 infrared detector and ASHRAE calculation methodologies (Persily and Grot 1985; ASHRAE 1981).

The authors have found that the achievement of design values of outside air can never be assumed without at least validating the most recent air balancing data and the current correct operation of the ventilating equipment.

Recommendations for Solutions

Once a good understanding of the current situation has been developed, recommendations can be made concerning the supply of adequate outsie air to the occupants. It is the opinion of the authors and several other researchers that many current building codes do not adequately specify the quantities of outside air that are necessary to provide conditions that will be considered acceptable in many of today's office environments.

Based on the current state of knowledge about this matter, the authors recommend the proposed ASHRAE 62-1981R minimum ventilation guidelines (ASHRAE 1986), which can be expected to be adequate for situations for which there are no known sources of contaminants in a space or building. Whenever the potential irritation of occupants in a confined space by identifiable sources, such as emissions from tobacco smoking, wet process photocopiers and large dry process copiers, or other potential point sources is suspected, special ventilation provisions are suggested. Typically, we recommend isolation of the source from the return air system and the use of a localized exhaust ventilation system as the most effective strategy.

PROBLEMS IDENTIFIED TO DATE

Based on indoor air quality evaluations that have been conducted by the authors since 1984 in office environments, the following are examples of problems concerning the delivery of outside air to the occupants of a building.

These situations relate to insufficient amounts of outside air being drawn past the dampers.

Situation A, Building Overpressured

In this example, the installed HVAC system in an office building was unable to deliver designed outside air and total air to the building due to installation errors. The HVAC system consisted of a prepackaged rooftop-mounted heat pump (with a field-installed economizer cycle), ducted supply, zone, and returns. Investigation of the control circuitry revealed that the building exhaust fans never operated due to improperly wired circuitry. This caused excessive positive pressure in the building (approximately .05" water, static pressure), which reduced airflows and resulted in difficulties in providing adequate free cooling air. In addition, a defective outside air damper control and the invalid assumption in the balancing report of a 10% outside air leakage rate lead to the outside air being "off" for at least a year.

Additional deficiencies of the system included an improperly adjusted warm-up cycle control, which may have also contributed to low outside air quantities by keeping the dampers closed after building occupancy. Poor zoning also led to local overheating due to the inability of the cooling equipment to respond to solar gains at the building's perimeter. The renovation of this building, prior to this occupancy, included the installation of unopenable windows that reduced infiltration, but did not include the installation of a continuous vapor barrier.

Further tracer gas testing has revealed that, in fact, even with the outside air dampers closed, approximately 1.25 air changes per hour of air was being moved through the building during relatively mild conditions. Thus, uncontrolled leakage due to a permeable shell is suspected of causing too much outside air to be supplied during severe winter weather, leading to extreme dryness and occupant discomfort.

The recommended solution includes the re-design of the system with adequate zoning to allow a response to solar gains and a complete checkout of the control logic for the HVAC economizer operation.

In addition, further investigation of the building's shell leakage through the use of infrared thermography and subsequent caulking is being investigated.

As this building has a high occupant density and was designed to recent energy codes, it is expected that the minimum position location of the outside air damper will need to be at approximately 30%, after the uncontrolled leakage is reduced.

Situation B, Lack of Minimum Open Position for OA Dampers

This situation has been observed for a number of reasons:

(1) There is the unsubstantiated belief that outside air dampers in the closed position still permit 10% to 15% leakage,

which may have been true at one time but is definitely not true now. Also, the control for a warm-up cycle was set at too high a temperature and so kept the outside air dampers closed for a longer interval in the morning than was intended, despite the fact that the building was occupied.

Situation C, Damper Control Out of Calibration

Another control-related problem occurred in a building where the mixed air sensor controller was out of calibration. Instead of seeking to cool the return air from 70F to 60F with the introduction of outside air, the sensor controller was seeking 70F and so kept the outside air dampers closed.

These following situations had sufficient quantities of outside air being drawn past the dampers, but there were problems with the delivery systems.

Situation D, Ducts Were Sized to be Too Small

This situation involved a building in which the installed HVAC equipment was unable to deliver design quantities due to flaws in the design. This constant volume HVAC system consisted of both ducted supply and ducted return, with no economizer mode. An original 20-year-old balancing report documented the HVAC system's inability to deliver specified outside air and total air quantities due to inadequate duct sizing. These deficiencies resulted in air quantities that were approximately 40% of design on the average with a range of 25% to 60%. Since this building was designed to standards developed before the energy crunch of 1974, the delivered quantities may be tolerable in this low-occupant density building. This situation has yet to be resolved, the project is ongoing.

Situation E, Leakage from Outside Air Supply Directly to Building Exhaust

In this office building example, the installed HVAC system was unable to deliver the designed quantities of outside air to the occupants. This was due to a number of factors including the fact that the use of the space was radically different from the original design for the building. The HVAC system was a constant volume, nonrecirculating type with a pressurized ceiling plenum supply, area exhausts, and no economizer mode. The original HVAC design was for residential apartment/condominium occupancy and was not sufficient for the ultimate office use. Aside from this change in space usage, tracer gas measurements revealed a complete inability of the pressurized ceiling plenum design with limited zoned exhaust ducting to distribute the outside air to the occupants. There was gross short-circuiting through duct leakage, which prevented the outside air that was being delivered to the ceiling plenum from ever reaching the occupants within the space, and the limited height of the ceiling supply plenum contributed to the distribution problem. All of these IAQ problems were aggravated by inadequate HVAC system maintenance and a lack of understanding by the maintenance staff of the system.

The recommended solution was to completely re-design the HVAC system for adequate supply and distribution or to dramatically alter the use of the building to very low occupancy or to the original residential purpose.

Situation F, Short-Circuiting of Supply Air to Return Air

In this school building, the installed HVAC system was unable to deliver designed outside air quantities to the occupants due to inadequacies in the installation details and design specifications. In this case, the HVAC system was of the constant volume type with an economizer mode. The distribution system consisted of a light troffer diffuser supply system with ceiling plenum return. Flow measurements revealed that short-circuiting was occurring at the site of the light fixture-diffuser outlet interface, allowing 30% to 50% of the supply air to short-circuit directly from the diffuser outlet to the return air plenum, without ever entering the occupied space. This problem occurred in a building of modern vintage (with high occupant density) such that this 30% - 50% reduction in delivered air quantities resulted in the delivery of approximately 5 cfm outside air per person or less; it is now widely accepted that 5 cfm outside air or less is inadequate to maintain acceptable IAQ unless special air cleaning is utilized. This problem was corrected by sealing the troffer diffuser to the light fixture through the use of a gasketing material and increasing outside air minimum set points.

CONCLUSIONS:

Through detailed investigation of actual airflows and operational parameters of the HVAC systems of numerous buildings, it has been shown by many researchers (including the authors) that an inadequate supply of "fresh" outside air has contributed to occupant discomfort and complaints of poor indoor air quality.

The inadequate supply of outside air may be caused by several factors, including outdated design standards (or building codes), the defective installation of the HVAC equipment, overloading by human occupancy densities much greater than the original design, or inappropriate operations and maintenance practices.

If ventilation inadequacies are expected to be identified and resolved, the assessments of outside air supply can only be accurately determined with defensible on site ventilation measurements, which must include an assessment of the actual outside air delivered to the occupants.

It is a challenge for today's HVAC and building designers to design systems that will not only minimize energy consumption but also provide occupant comfort. These systems must be installed correctly and maintained to operate as designed.

Building owners and occupants must be made aware of the limitations of the design, so that systems do not become overloaded, or inappropriate air contaminants added.

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BUILDING SYSTEMS: ROOM AIR AND AIR CONTAMINANT DISTRIBUTION

SYMPOSIUM TOPIC: VENTILATION MEASUREMENT AND VENTILATION EFFICIENCY

PAPER TITLE: DETERMINATION OF OUTDOOR AIR QUANTITIES DELIVERED

BY: DAVID W. BEARG, P.E. & WILLIAM A. TURNER, P.E.

ABSTRACT

As part of an indoor air quality investigation protocol, frequently the quantities of outside air being delivered to the occupants of a building is identified as in need of assessment.

Methods frequently utilized for this task include tracer decay assessments and the measurement of indoor CO^2 levels, assumed to be generated by the occupants. This paper presents data from buildings in which both CO^2 measurement and tracer gas decay were performed separately or simultaneously in order to assess the amounts of outside air reaching the occupants.

The methods utilized will be explained and discrepancies in the results of the two methods will be discussed. Discussion of "ventilation efficiency" - (the delivery of outside air to the occupied space vs. the quantity of outside air brought in at the air intake) are presented.

Protocols are suggested which are expected to minimize errors in the correct interpretation of the results and further research needs are discussed.

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INTRODUCTION

As part of an indoor air quality investigation protocol, the major focus is frequently a determination of the quantities of outside air (OA) actually being delivered to the occupants of a building. This quantity of OA being delivered to the occupants is a function of both the quantity of OA taken in at the OA dampers and the effectiveness of the HVAC system in delivering this air to the people. In this paper, it is the ratio of the air quantity entering the OA dampers and the outside air quantity delivered to the occupants that is referred to as the "ventilation efficiency." It is desirable to know this ventilation efficiency in order to determine if improvements in delivery efficiency are warranted. Measurement of both the delivered quantities of outside air and the efficiency of the ventilation system are especially important in light of various protocols for design options that are contained in proposed ASHRAE Standard 62-1981R, "Ventilation for Acceptable Indoor Air Quality." The two principal tools currently utilized for evaluating the quantity of outdoor air delivered to the occupants of a building are the measurement of carbon dioxide (CO^2) concentrations and the measurement of tracer decay rates using sulfur hexafluoride. These methods have also been utilized by the authors and other researchers to assess the efficiency of outside air delivery. Each of these techniques has its own set of advantages and disadvantages, but the performance of the two together tends to cancel out some of the weaknesses. Therefore, whenever possible, the authors have performed both procedures side by side to maximize the precision and the accuracy of the results. This paper discusses some of the issues raised by these comparative results.

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METHODS

<u>Discussion of Information That Can Be Derived From Carbon Dioxide Testing</u>

The ventilation rate determination using CO^2 measurements compares the indoor concentrations with outdoor concentrations and assumes that the increase in indoor values is due to the buildup of CO^2 expired due to respiration by the building occupants. This relationship is detailed in Appendix D of the ASHRAE Standard 62-1981 (ASHRAE 1981). The advantage of this technique is that relatively few measurements obtained during the peak afternoon occupancy period can provide a determination of the quantity of ventilation air being provided. These results express the ventilation rate in terms of cubic feet per minute of outdoor air per person (cfm of OA per person), which are then directly comparable with the recommended minimum OA quantities published in the ASHRAE Standards 62-1981 and 1981R (ASHRAE 1981, 1986).

Although this comparison is very useful, it is not always sufficient for the scope of many evaluations. Most often, in order to compare the quantity of OA delivered with the quantity entering the HVAC system, the quantities need to be expressed in terms of just cfm of OA. The cfm of OA at the air intake is determined by flow measurements, while the delivered quantity is the product of the CO^2-derived cfm/person value and the total number of people present at the time of the test measurements. In actual testing situations then, it can be important to know exactly how many people are actually present in the space at the time of the CO^2 measurements. For populations up to 30 or so people this

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rarely represents a problem, but for 300 people or so, the ability to count the number of people is much more difficult. Techniques for estimating this population are, of course, possible. As part of building surveys, we have measured not only CO^2 concentrations, but potential number of people per office and actual number of people per office. Then, assuming that the 20% of the "random" offices surveyed are typical of the rest of the building, we can estimate the total building population. This then yields the OA quantity in terms of cfm.

Dividing cfm of OA value by the area involved yields another important evaluation criterion, cfm of OA per square foot. Corporate ventilation standards are often expressed in terms of minimum quantities of cfm of OA/ft^2.
For typical office environments (as defined by the ASHRAE Standard 62-1981R), with 7 people per 1,000 square feet of area, the proposed ASHRAE minimum ventilation rate value of 20 cfm of OA per person is equivalent to 0.14 cfm of OA per square foot of office space. Total air delivery rates, as contrasted from outdoor air delivery rates, are typically around 1.0 to 1.2 cfm per square foot of office space.

In addition to expressing the ventilation rate in terms of cfm of OA per person or per square foot, the ventilation rate can also be expressed as the number of air changes per hour (ach). It is this expression of the ventilation rate that most accurately represents the rate at which air contaminants are diluted and removed from the space. This is also the format for the results from tracer decay ventilation testing. For the typical office, with 20 cfm/person, 7 people per 1,000 ft^2 and total ceiling heights of about 10 feet, the ventilation rate would be 1.0 ach. Factored into this calculation is the empirical result, from tracer gas studies, that the net effective volume can be estimated as 85% of the gross volume of the space. These distinctions in expressing ventilation

rates become especially important in atypical spaces. For example, in one evaluation of a complaint building with total ceiling heights of 18 feet, utilizing the CO^2 approach, data were collected that yielded a peak average indoor concentration of 713 ppm with 425 ppm outdoor conditions, yielding a ventilation rate of 36.5 cfm of OA per person. For the population estimate of 300 people, this represented 10,950 cfm of OA. Tracer gas testing conducted at the same time yielded a value of 0.65 ach. When this ach was applied to the area of 60,000 ft^2 and an estimated effective volume of 972,000 cubic feet, it yielded 10,530 cfm of OA. Both of these values can be expressed as 0.18 cfm of OA/ft^2. The question remains, however, as to which criterion is the one to use in recommending a ventilation rate sufficient to prevent complaints of inadequate ventilation. In this situation, it is the ventilation rate expressed in terms of ach that is most directly concerned with the rate at which air contaminants such as environmental tobacco smoke (ETS) is diluted and removed from this space.

Research Need:

The determination of the most appropriate ventilation goal and the criteria of meeting that goal; i.e., cfm/PERSON, cfm/ft^2, or ach can have a profound effect on the resulting air quality in the space and the amount of outside air needed to provide acceptable air quality. It is in this area where more research is required, especially related to the efficiency of delivery of the outside are to the breathing zone, or occupied zone.

Measurements of CO^2 concentrations at different indoor locations can point out localized variations in ventilation rates throughout the building.

Measurements comparing the concentrations in the supply ductwork and the return ductwork as well as the occupied spaces can characterize the ratio of

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outdoor air to recirculated air leaving the HVAC equipment. Measurements obtained in the early morning can document the persistence of air contaminants from the evening before where the building occupancy extends beyond the operation of the ventilation system. Early morning measurements can also document the impact of air contaminants from parking garages.

LIMITATIONS OF CARBON DIOXIDE TESTING ALONE

The measurement of CO^2 concentrations has also been used by the authors to detect the presence of inadequately vented combustion appliances; e.g., a poorly vented gas-fired hot water heater in a university building. The interference of CO^2 from sources other than the unaccounted for presence of the products of combustion can lead to very erroneous ventilation rates. This was reported in the case of an investigation of a warehouse/showroom that was heated with direct-fired gas heaters. Therefore, in order to trust the results from a CO^2 analysis, it is necessary to be certain that there are no products of combustion entering the building or mechanical systems.

Another disadvantage of this approach is the requirement for a sufficient density of people in order to obtain a measurable increase in indoor concentrations as compared with out-door concentrations. The extreme example of this is when we have been called upon to evaluate a building that has been evacuated and is devoid of the products of human respiration. This situation then requires the use of tracer testing for a ventilation rate evaluation.

METHOD

Discussion of Information That Can Be Derived From Tracer Gas Testing

As already mentioned, the tracer decay testing approach yields its results in terms of ach. This approach is based on measuring the rate of decrease of a tracer, such as sulfur hexafluoride, that has been uniformly dispersed throughout the space under investigation. If the space under investigation is behaving as a single, well-mixed chamber, the measurements will yield a straight line on semilog plot. The slope of this plotted line is equivalent to the ventilation rate and can be expressed in terms of ach. Calculations are based on the relative changes in tracer concentration and do not require an absolute calibration of the analytical equipment.

Many office spaces, with typically in excess of 85% of the air being recirculated, result in at least the supply air being well mixed. Distribution inefficiencies, however, may result in stagnant zones in the occupied spaces which are not well mixed. Distribution inefficiencies, such as a well mixed zone near the ceiling and stagnant areas below will also complicate the analysis by causing a deviation from the single zone assumption.

The actual shape of the tracer decay plot provides useful information to characterize the behavior of the ventilation system. Slopes that deviate from linearity (on a semilog plot) indicate variations from the well-mixed case; slopes with the appearance of an increasing slope with time are representative of displacement or plug flow, while slopes with the appearance of a decreasing slope with time are representative of incomplete mixing and zones of stagnation. As with the CO^2 approach, the quantity of OA may need to be expressed in terms of just cfm of OA in order to evaluate the performance of the HVAC system. This value can be determined by multiplying the tracer decay

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derived ventilation rate, in ach, by the volume into which the tracer was dispersed. This mixing volume for the tracer is referred to as the "effective volume" and can be determined from procedures that require a very precise determination of the rate of tracer release and the absolute quantity of the tracer measured.

Research Need

Most building investigations do not have the budgets to support the precise determination of the effective volume of the tracer dilution, thus an estimate based on the gross volume is more typically used. Our experience indicates that estimates of effective volume that are 85% of the gross volume yield reasonable results. This is an area where more research is required.

COMPARATIVE ADVANTAGES AND DISADVANTAGES OF EACH APPROACH

The authors have been known to say that "most buildings are similar but each building is unique." It is some of these unique situations that point out the strengths and weaknesses of these two investigatory approaches. In one example, one part of a building was being impacted by air contaminants being generated in another part of the building. The air flow relationship was such that the air being delivered to the space being evaluated had already traveled through another occupied part of the building. Tracer decay testing alone performed in this space would fail to take this into consideration and yield an outside air ventilation rate much higher than is actually being provided. The CO^2 approach, however, by measuring the concentration in both the supply air and occupied space would provide a more accurate assessment of the outside air ventilation rate (in an occupied building). In this situation, the evaluation included several locations in a building complex that consisted of two adjacent buildings that had been interconnected. The direct release of the tracer into

the occupied area of the smaller of the two original buildings, with 4,100 ft^2 per floor (the adjoining building has 16,500 ft^2 per floor), yielded a linear decay rate equivalent to 4.3 air changes per hour. For the estimated volume of this space, this represented 2,320 cfm of OA. The CO^2 test results, however, based on 700 ppm indoors and 400 ppm outdoors with 17 people present in the space yielded a ventilation rate of 35 cfm per person or a total of 595 cfm of OA. An additional tracer test of the same study area, but with the tracer being released into the HVAC system located in the larger building three floors above, yielded a tracer decay rate equivalent to 1.1 ach. This 4,100 ft^2 area had a total ceiling height of 9.3 ft leading to a gross volume of 38,100 ft^3. Using the previously mentioned relationship of the effective volume for occupied office spaces being 85% of the gross volume, the net volume is 32,400 ft^3 and the 1.1 ach yields an effective ventilation rate of 594 cfm of OA, which is the same result as that from the CO^2 calculation.

This example also points out that with both assessment approaches, it is crucial to be familiar with the design of the HVAC system as well as its intended modes of operation and the need to be aware of any changes in the operation of the system that could affect the quantity of OA being delivered throughout the testing period.

Since it is easier to perform the CO^2 measurements than to perform the tracer analysis, the CO^2 technique will be the more likely analytical procedure of choice. Specific reasons for performing both procedures are situations where there are only low densities of people in the space of concern, or it is difficult to assess the number of people present in the building, or if it is important to understand the air movement patterns among adjacent zones of the building.

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DIAGNOSTIC ASPECTS OF SICK BUILDING SYNDROME: COMPREHENSIVE IAQ/HVAC SYSTEM DIAGNOSTICS FOR BUILDINGS WITH ELEVATED IAQ COMPLAINT LEVELS

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ABSTRACT

During the past decade, there has been an increasing awareness of health concerns regarding exposure to low levels of pollutants in the indoor office environment. A large number of buildings have been studied and the results reveal a variety of similar complaints and symptoms. These complaints often center around issues of thermal discomfort or health related complaints (irritation of the eyes, nose, throat and lungs). Unfortunately for the building manager, the causes of these complaints and health concerns are unique to each building. Recently Dr. Ole Fanger, a noted researcher of the Technical University of Denmark, has presented information quantifying air pollution as it is perceived by humans indoors and outdoors. The results of Dr. Ole Fanger's work have suggested that in many buildings the HVAC system is perceived as significantly contributing to the causes of the occupants complaints.

It has been the experience of the Authors and other researchers that Industrial (OSHA) type pollutant surveys that are often conducted by in-house staff or consultants, typically provide little insight because the results are often reported as below OSHA standards or below minimum detectable standards. This often leaves the occupants with an even greater sense of frustration.

Evaluation and measurement techniques have been developed and utilized that are capable of identifying the causes of the occupant complaints. Often equipment is employed that is 10 - 100 times more sensitive than occupational type surveys would dictate. The purpose of these low level measurements is to evaluate what is actually happening to the air within the building - not just whether the air quality meets certain OSHA requirements. These evaluation techniques must include a very comprehensive evaluation of what the building's HVAC system is or is not accomplishing for the occupants.

This paper presents an evaluative framework that has proved very useful in buildings in which there is no identifiable suspected source of air contaminants, yet there are complaints; and in situations where there is an identifiable suspected source, but unknown pathways of transmission. To date we have conducted evaluations in over 3 million square feet of office, research, and institutional type space utilizing this basic approach.

Once a good understanding of the history of complaints, current delivered ventilation rates, pollutant levels, air flow patterns, pollutant sources and control equipment has been developed, corrective actions can be developed and implemented, if warranted.

INTRODUCTION

In the process of conducting indoor air quality investigations in office type buildings, the authors have identified several important areas that need to be evaluated. These include, but are not limited to, (1) the history of the occupant complaints, (2) an evaluation of the distribution of "fresh" outside air (OA) to the occupants, and (3) an inventory of the sources of potential irritants located both inside and outside the building. This paper focuses on the overall techniques utilized in conducting these investigations: the evaluation of the actual amount of outside air being distributed to the occupants has been described elsewhere. [1]

History of Occupant Complaints and Building Operation

It has often proven critical for the investigator to develop an understanding of the history of occupant complaints and previous attempts at corrective action. In order to gather this information, it is useful for the investigator to meet with all parties involved, especially, but not limited to, managers, employees, and individuals responsible for the building operation.

Developing a history of the occupant complaints is necessary in order to develop a hypothesis as to whether the complaints are related to indoor air quality, temperature/humidity control, poor lighting, job stress or some combination of the above.

The performance of effective indoor air quality (IAQ) evaluations depends on the ability to understand the interactions of the variables affecting IAQ. These variables include the effective ventilation rates within the building (quantities of "assumed fresh" outside air delivered to the occupants) and the introduction of air contaminants from either within the building, outside the building or from within the mechanical or unoccupied spaces of the building.

In determining the amount of outside air made available to the occupants, we have identified three important questions:

1. What was the space originally designed to be used for? (i.e.,what was the original intent of the design, based on drawings, specifications and design quantities, or does anybody know?)

2. What is the space currently being used for? (i.e., based on inspection and defensible measurements, what is really happening there, loads, ventilation, etc?)

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3. What do we want to be happening?
(i.e., based on current and planned space usage and the current and evolving guidelines concerning the quantities of "fresh" outside air that are considered desirable to deliver to occupants, what should be happening?)

Original Design: The determination of the original intent of a design (i.e., what was supposed to be happening in this space) may be as simple as finding the original design drawings and specifications and extracting information. For older buildings, which have changed owners several times, this can be a tedious chore and success may be determined by luck, not by skill alone. The task is often easiest when the building is owner-occupied, and this original owner is the client.

Historic and Current Situation: The determination of what is currently happening in this space (with regards to the HVAC system and air flow patterns), and possibly in the recent past, is a matter of meticulous inspection, testing, and measurement. By inspection and communication with the owner, one can determine the building's actual current occupancy, loads, space utilization, and frequently, evidence of the current HVAC preventative maintenance. Historical conditions can only be inferred from the situation as found.

The actual amounts of outside air being delivered to a space, can be evaluated only by testing and measurement. This testing must, at a minimum, include the inspection and testing of all controls that affect the delivery of ventilation and outside air to the occupants and the actual measurement of the quantities of outside air and/or total air (TA) in question.

The authors have found that the achievement of design values of outside air delivery can never be assumed without at least validating the most recent air balancing data and the current correct operation of the ventilating equipment.

ASSESSING ACTUAL BUILDING VENTILATION RATES

The concept of Effective Ventilation Rate (EVR) refers to the ability of the ventilation air to remove air contaminants from the occupied spaces in the building. One procedure for determining the effectiveness of the ventilation system involves the measurement of carbon dioxide (CO_2) concentrations throughout the building. In addition to measuring CO_2 levels in the occupied spaces, measurements should be obtained of the concentration of CO_2 in the supply air, and outdoor air in order to assess the proportion of outdoor air coming from the mechanical HVAC system versus the building enclosure infiltration. Testing should be thorough enough to note variations, both by time and by location.

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Peak values of CO_2, for instance, typically occur in late morning and mid-afternoon. If it can be determined that the occupants are the only source of the CO_2 in the building (that is, that there are no combustion sources venting to the occupied spaces), then the resulting CO_2 concentrations can provide a measure of how well the ventilation system is diluting and removing air contaminants generated within the building. [2]

This approach of determining the effective ventilation rate is especially useful when the air contaminants of concern are being generated within the occupied spaces since, if the CO_2 is not being effectively removed and its concentration is building up, then similar conclusions can be made about other components of the indoor air as well. The specifics of converting CO_2 concentrations to quantities of outside air delivered (expressed in CFM/person) are presented in the American Society of Heating, Refrigerating and Air Conditioning Engineers, Inc. (ASHRAE) Handbook of Fundamentals. [2]

Techniques for conversion of these CFM/person values to CFM values has been discussed in detail in a paper presented by Turner and Bearg. [3]

MECHANICAL SYSTEMS

For mechanical HVAC systems, a useful procedure for determining an outside air intake rate in a mechanically ventilated building, is to perform an enthalpy balance at the mixing box adjacent to the outside air dampers. The term "enthalpy" refers to the internal (sensible plus latent) energy in a mixture of moisture and air. Knowing the enthalpy of the outside air, the return air and the resulting value for the combination of these two air streams, you can determine the relative proportion of outside air to return air that are being mixed together at the time of measurement. Accurate, stable, calibrated sensors are needed when making this assessment.

This technique, however, only assesses the outdoor air introduced into the mechanically driven air handling system. A shortcoming of this approach is that it does not take into consideration outdoor air that may be introduced by infiltration. Also, merely determining the amount of outdoor air introduced into the mechanical system will not indicate how much of this outside air may be wasted, (not actually reaching the building's occupants), because of duct leakage, short circuiting or other system inefficiency.

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AIR MOVEMENT PATHWAYS AND VENTILATION EFFICIENCY

The determination of a building's air movement pathways and the efficiency of the ventilation system can most readily be accomplished by tracer gas testing, or a combination of tracer gas and carbon dioxide testing. Releasing a tracer such as sulfur hexafluoride near the site of an identified source, with a subsequent analysis of samples collected throughout the building, is the basic technique which will identify transmission pathways. [4]

This technique provides an assessment of how rapidly that quantity of tracer reaches other locations, as well as a determination of the pathways of air movement through the structure.

Another technique for determining air movement pathways is the use of air current tubes. These devices produce a neutral density visible cloud that will follow the air streams into which they are released.

ASSESSMENT OF BUILDING CONTAMINANTS

After assessing the effective ventilation rate for removing air contaminants from the occupied spaces and the pathways of air movement into and through the building, the next step is to assess the presence of air contaminants themselves. First, it must be recognized that the sources of these air contaminants can either be from within the occupied spaces of the building, from outside the building or from within the mechanical system or other unoccupied spaces in the building. Since the determination of the effective ventilation rate provides a measure of how effectively pollutants generated from within the occupied spaces are being removed from these spaces, it is useful to perform an emissions inventory to determine the possible sources of contaminants.

SOURCES WITHIN THE BUILDING

The following is a listing and discussion of some of the typically more important indoor air contaminants:

(1) Particles: The first source of particles to consider is the presence of smoking activity. Qualitative assessments can, of course, be made by simple observation. Quantitative assessments can be based on measurements of concentrations of respirable particulate matter (RSP) or nicotine. Approximately 95 percent of the smoke generated from cigarettes is in the respirable range. Care must be taken in interpreting these results since cigarette smoke may not be the only source of RSP in a given area. Portable equipment currently exists for near real-time determinations of RSP concentrations, thus allowing walk-through surveys to be conducted. In evaluating the impact of tobacco

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smoke, two mechanisms for elevated concentrations of RSP have been observed: local generation and transport by the HVAC system from other locations also served by that system. If tobacco smoke is determined to be a cause of concern, methodologies of providing smoke-free air are available. [5]

Very large dry process photocopying equipment has also been determined to contribute fine black carbon particles to the indoor air, if the machines are not exhausted to the outdoors.

(2) Combustion gases: The inventory of potential sources must include all combustion gases. Combustion oxides (often respiratory irritants) are generated by combustion processes. These oxides include carbon monoxide, carbon dioxide and nitrogen dioxide. Real-time portable monitors are available for all three oxides and passive integrating samplers are available for carbon dioxide and nitrogen dioxide.

(3) Ozone: The potential for ozone may be related to the quantity of mis-adjusted electrostatic copiers in the building or other high intensity UV processes. Real-time portable ozone monitors may be used to conduct surveys if suspect processes are in use, and often an odor is prevalent near the source.

(4) Biological sources: The presence of excessive mold, fungi, and bacteria can often be related to evidence of localized high humidity conditions (in excess of 60 percent relative humidity or condensation on a surface), which fosters the growth of these organisms. This assessment should include the occupied spaces and any humidification equipment or mechanical systems. Specific types of equipment implicated in past evaluations as sources of potentially unhealthful microbial aerosols include water spray systems, humidifiers that use recirculated water, cold mist vaporizers, poorly maintained AC fan coil units, flooded carpets, and leaky roofs. [6]

Sampling for the concentrations of viable organisms in occupied spaces typically involves drawing the room air across an agar medium, or bulk sampling of suspect carpets and duct liners. Analysis of this culture plate involves incubation and subsequent identification and counting of the number of colonies formed. Other biological indicators may include a presence of insect parts. A procedure which involves microscopic examination for insect hairs or body parts may be helpful in identifying the source.

(5) Organic chemicals: The complexity and diversity of the organic chemicals introduced into many indoor environments can make assessments of these compounds difficult and air cleaning for their removal impractical. Hence, designs have typically relied on the effectiveness of the general ventilation system, or a well designed local exhaust system, for the removal of the off-gas contributions from building materials and office equipment.

More recently the specification of low emission products has been utilized. Real time survey monitors are available for identifying many potential sources and sophisticated, expensive analyses such as GC/MS or MS/MS can be performed to determine the specific components of the organic soup in modern office buildings, if a particular agent is suspected. Passive integated samplers are available for many compounds.

(6) Fibers: The presence of fibers, including fiberglass and asbestos, may be related to the presence of friable acoustical or insulation materials often located within the ceiling plenum of a building or in mechanical rooms. If there is frequent construction activity, water leakage, and transmission of these materials to other areas of the building, there may be increased risk of exposing the whole building to these air contaminants.

SOURCES FROM OUTSIDE THE BUILDING

The following is a detailing of air contaminants that typically originate outside of the occupied spaces in the building and are drawn into the building either through the mechanical system or through infiltration.

During the heating season, buildings usually behave like chimneys. This "thermal stack" effect is created by the escape of buoyant heated air at the upper levels of the building, which creates negative pressures capable of causing infiltration at the lower levels of the building, unless specific steps have been taken to eliminate the effect. Therefore, because of this induced suction, the following items can be identified as potential sources of air contaminants to the building:

(1) Radon and other soil gases: The potential for radon problems prior to construction, can only be crudely assessed by determining the radon content of the soil geology, sources of water and building materials. After construction, very definitive assessments can be provided by intrgrating detectors or real-time monitors. The release of radon decay products into a building is most often related to the infiltration of soil gas, or outgassing from ground water supplies, both of which can easily be determined and mitigated. Hydrocarbon soil gases from leaking underground fuel tanks and landfill decomposition products have also been known to cause problems.

(2) Loading docks: They are considered as potential sources of air contaminants for many buildings. The combination of idling diesel vehicles and penetrations at the loading dock, under the influence of typical negative pressures, will draw pollutants into the building's interior, unless the area has been designed to prevent this from occurring.

(3) Parking lots and roadways: They, too, are considered as potential sources of air contaminants for some buildings because of the accumulation of carbon monoxide and carbon dioxide from idling vehicles. This is especially true for multilevel parking facilities in congested areas. These may be considered large area sources for pollutants such as carbon monoxide, and other combustion irritants.

(4) Cooling towers: Poorly maintained towers have been known to be a source of biological growths that may cause infections in people under unusual circumstances.

(5) Localized exhaust systems: These systems (fume hood exhausts, toilet exhausts, print room exhausts, etc.) should also be evaluated. If their points of emission are close enough to air intakes or infiltration sites, or the emission is captured in the wake of the building, the contaminants will be drawn in with the outside air. In evaluations of this type, algorithms have been developed and published for estimating air intake contamination from nearby exhaust vents. [7]

The parameters for evaluating their impact on IAQ include the capture efficiency, room pressure relationship, and the exhaust discharge parameters. The evaluation of localized exhausts, such as fume hoods or process emissions, cuts across the distinctions between the above items and, therefore, requires a "systems approach" in order to account for several interrelated factors.

SOURCES RELATED TO THE BUILDING MECHANICAL SYSTEM

(1) Location of air intakes: The location of air intakes can contribute to the contamination of indoor air quality if they permit reentry of emissions from the building itself or from other sources.

(2) Humidity control: Any humidification or dehumidification device involving recycled and/or standing water is subject to stagnation and is a potential source of contamination from microorganisms, including various fungi.

(3) Location of supply and exhaust registers: Register locations contribute to the determination of ultimate indoor air quality by their varying abilities to remove contaminants from the occupied zones. If both supply and exhaust registers are located in the ceiling or in the same wall, short circuiting may occur. This may allow contaminants to remain undiluted within the stagnant zone. However, a floor-supply/ceiling-exhaust geometry in combination with a buoyant contaminant, or high supply low return, can help drive contaminants from a space.

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(4) HVAC preventive maintenance: Poor historic maintenance of the air filters and condensate drip pans may contribute to conditions favorable to microbial growth within the HVAC system. Lack of proper filter maintenance may allow considerable organic substrate to accumulate within the air handling unit and down stream. Lack of proper drip pan maintenance or poor drainage detail may allow for the buildup of a microbial soup, supporting the growth of organisms known to elicit allergic responses in humans, or in some cases known to be pathogenic to humans. Bulk sampling of accumulated material and subsequent analysis by experienced microbiologists can be performed.

BUILDING HEIGHT

Building height is also included as a factor affecting indoor air quality because the taller a building, the greater the potential for pressure which the air distribution system must overcome in order to distribute air throughout the structure. In many buildings with roof-mounted equipment, the return air riser behaves like a chimney, drawing more air than intended out of the lower levels. With improper design, the return fan output overtakes the supply fan, and the building air pressure becomes negative to the outdoors on the lower floors and over-pressurized on the upper floors. If the air pressure in a space is greater than what the supply fan can deliver, the occupants on the upper floor levels then receive no air.

Another serious ramification of this problem is that the return air volume can exceed the supply fan volume. The outside air opening, thus becomes a building exhaust port; and even though the outside damper may be wide open, no fresh air is introduced.

SUMMARY AND CONCLUSIONS

The presence and location of various potential sources, both inside and outside of the building, can be confirmed by observation and measurement if necessary. In order to determine how the ventilation system or systems of a building are performing under various operating conditions which occur, and how the changes in ventilation rate would effect the pollutant concentrations, it is necessary to use sophisticated measurement approaches. The evaluation of a ventilation system requires the measurement of at least one component of the gas stream. Options for this evaluation include compounds that are normally present in the occupied spaces or a compound that is specifically introduced into the occupied spaces for the purpose of this evaluation, such as sulfur hexafluoride.

If the evaluation of ventilation rates and the determination of air movement pathways are not sufficient to deduce the source of air contaminants, further specific measures of sources and contaminant concentrations may be employed.

To date we have conducted evaluations in over 3 million square feet of office, research, and institutional type space utilizing this basic approach with good success at a resolution of the problem.

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RADON EVALUATION, DIAGNOSTICS, MITIGATION AND NEW CONSTRUCTION: STATE OF MAINE OCCUPIED AND FUNDED BUILDINGS

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ABSTRACT

For many years, there has been a concern for exposure to radon gas and its progeny in Maine. This concern was first identified as being predominantly associated with exposure to the use of well water containing elevated levels of dissolved gas and radioactive minerals. In more recent times, the concern has also been associated with exposure to the influx of soil gases. This paper presents a summary of the scope and available results of current programs being conducted by the State of Maine with regard to the identification and mitigating of elevated levels of radon in state occupied buildings and schools and the subsequent planning for mandatory radon-resistant new construction features in all state funded projects as needed. These programs are part of a wider comprehensive State of Maine program that addresses indoor air quality in state occupied or funded buildings.

INTRODUCTION

Radon and its progeny have been a concern in Maine since the early 1950's and 1960's when elevated levels in drilled wells were studied by several researchers. (1) Smith In more recent times, researchers at the University of Maine at Orono and Maine Medical Center (Portland) have focused on the exposures to occupants of homes with elevated indoor levels and on corrective measures for drilled wells. (2) Lanctot, (3) Hess, (4) Lowery

Recognizing the possible health concern for state employees and school children, the State of Maine, Division of Safety and Environmental Services (DSES), Division of Health Engineering (DHE), and Department of Education (DE), embarked on a joint program during the winter/spring of 1989 to identify elevated indoor exposures in existing facilities, caused by soil gas infiltration. Due to limited funding available in the 1989 fiscal year, the program was first directed at surveying a portion of the state occupied buildings located in expected high risk areas.

During the spring of 1990, with an influx of bond funding and additional support from US EPA federally supported programs, a large effort was undertaken to survey all earth contact classrooms in all schools.

Based on preliminary data from the state-occupied buildings and school surveys, and emerging national data from the US EPA, the State has also begun efforts to prevent future problems in state funded new construction by implementing "draft" mandatory Radon Resistant New Construction Standards in areas known to have elevated radon levels in unprotected buildings. (5) SOM

STATE OCCUPIED BUILDINGS, SURVEY AND RESULTS

In order to maximize the likelihood of identifying buildings with elevated radon levels in state owned or leased buildings with the limited funds that were initially available, buildings were selected for survey using several criteria. Out of 1200 buildings of various types, 200 were selected for initial screening. Their selection was based on their proximity to geologic formations known to be associated with sources of radon laden soil gas, a review of indoor radon sampling data available from the State of Maine, Department of Human Services Public Health Laboratory (a map was developed), the expected population exposure at the site, and in some cases a desire to obtain data from areas of the state where few measurements were available.

Results of the initial screening with diffusion type charcoal canisters revealed sixteen sites out of 180 with readings over 370 Bq/meter cubed (10 pCi/l). Follow up of four of the highest levels revealed one site with an open sump and three sites where detectors had been placed near floor cracks. Subsequent re-testing of exposures in these three sites revealed only one to actually be elevated. All of the sites with elevated levels will have further evaluations done in 1990 as funding is available. Further testing is planned at additional sites in 1990-1991 as funding is available. Results of independent co-located testing, utilizing a different type of detector, revealed good accuracy of the diffusion type detectors utilized.

SCHOOLS SURVEY

During March of 1990, all earth contact rooms utilized for instructional purposes, in all schools supported by state funds, will be screened utilizing commercially available charcoal detectors with liquid scintillation analysis. (6) Godzins A quality assurance program of paired sampling and independent checks with State of Maine Public Health Laboratory supplied diffusion barrier type detectors will be conducted as part of the program. (Unpublished protocol derived from the literature.)

After much discussion, a decision was made that the initial screening survey of over 10,000 rooms would be conducted during unoccupied weekends, (during the heating season), with any HVAC systems in the normal weekend mode. This approach was taken, as opposed to the current recommended US EPA protocol of switching to weekday operation of the HVAC system on the weekend, in light of the difficulties posed of ensuring that all schools would be operated on a weekday HVAC schedule for the test weekend. There are plans for follow-up testing in schools with elevated levels that will include weekday testing during the normal occupied mode in order to determine exposure assessment.

Once the results of the survey are available, schools will be prioritized for further evaluation, radon/HVAC diagnostics and mitigation as needed. It is currently expected that the State will request to participate in the US EPA sponsored "School Evaluation Program" once elevated school exposures have been identified.

PLANNING FOR NEW CONSTRUCTION

In the spring of 1990, the Bureau of Public Improvements, Division of Safety and Environmental Services, developed a draft Mandatory Radon-Resistant New Construction Standard. This effort is based on preliminary data from current state surveys, emerging national data, and a desire to reduce the likelihood of elevated radon levels in newly constructed schools and state funded buildings.

The draft proposed standard will require all new construction to meet an indoor standard of 74 Bq/meter cubed (2 pCi/L) or less, upon testing after occupancy. Guidance for minimum requirements for radon-resistant construction practice is being developed for areas known to have elevated soil gas levels. Currently, the need for the minimum practice is to be based on radon data from historic testing in earth contact rooms of adjacent unprotected structures and from passive soil gas "Bore Hole" testing data at an undeveloped site. It is acknowledged that the passive "Bore Hole" test method for an undeveloped site is experimental; however, the data can easily be collected during normal site geologic evaluation for structural design.

Features of the new construction guidance include, but are not limited to, the installation of a porous aggregate, a reinforced vapor barrier, sealed expansion joints and other earth openings, and passive vent stacks. Provision for making the passive soil gas venting system active, if needed, are required at sites with known strong radon sources.

Current estimates by the State of Maine suggest that the additional cost of the required radon resistant features are expected to increase construction costs by less than $4.30 per sq. meter ($0.40 per sq. ft.)

The law requiring mandatory Radon Resistant Construction is currently proposed to take effect in July 1990.

DISCUSSION AND CONCLUSIONS

All of the programs presented in this paper are part of an overall comprehensive Indoor Air Quality Program administered by the various Departments within the Bureau of Public Improvements (BPI) and Department of Human Services (DHS). Other elements of the programs include legislative sponsored programs designed to limit exposures to asbestos fibers and to ensure adequate minimum ventilation in state occupied buildings and schools. All newly constructed or renovated state occupied buildings (owned or leased) and schools are required to be designed to provide the occupants with minimum ventilation levels as specified by the ASHRAE Std. 62-1989 "Ventilation for Acceptable Air Quality." In addition to the current radon survey, a survey of ventilation (Carbon Dioxide levels) in portable (mobile) classrooms is planned for early 1990.

The Department of Human Services, Division of Health Engineering has been designated the lead agency in Maine to coordinate radon related activities with US EPA. Several training courses have been co-sponsored with the vocational technical colleges to train potential radon mitigators. Maine's participation in the US EPA sponsored state-wide radon survey in homes indicated an average screening value of 152 Bq/meter cubed (4.1 pCi/L) with 30 percent greater than 148 Bq/meter cubed (4 pCi/L) of 839 homes tested. Recently, comprehensive legislation has been passed requiring the registration of individuals or firms offering radon testing and mitigation services and reporting of test results and zip codes to the Division. State-wide screening of residences is encouraged but not envisioned to be done by the State of Maine. With the recent influx of federal support for staffing, educational programs and a follow-up of classrooms which indicate elevated values of radon from the screening tests will be performed.

The Division of Safety and Environmental Services has an evolving system of response to indoor air quality (IAQ) complaints supported by the Department of Human Services (Health Engineering and the Public Health Laboratory) and the Department of Labor (Labor Standards). The program includes active cooperation with State Employee Labor organizations and the use of consultants for in depth evaluation (assessment) and remediation of complaints. The Complaint/Response has already encountered radon as a complaint, as well as a variety of more typical IAQ issues. Support for DSES activities and utilization of services are clearly on the increase. The efforts described here can therefore be considered the foundation of an effective IAQ program in Maine State Government.

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Building HVAC/Foundation Diagnostics for Radon Mitigation in Schools and Commercial Buildings : Part I

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Abstract:

During the summer of 1989 US EPA embarked on the School Evaluation Program. In its initial stages this program has focussed on performing diagnostics on schools which have been determined to have elevated radon levels. To date these diagnostics have been performed in several diverse geographic areas of the United States, with more activity scheduled throughout the remainder of 1989 and 1990. The evaluated buildings have also been, and will continue to be utilized for teaching purposes for regional school personnel. Early in the program it was observed that schools, like commercial buildings, are typically designed with a more extensive and sophisticated heating, ventilating and air conditioning (HVAC) system than homes. The air handling system has a high likelihood of affecting the pressure relationship of the building envelope, which affects the radon gas entry rate. Through the implementation of a diagnostic protocol which includes foundation and subslab testing along with HVAC system testing, much has been learned concerning the dynamic interaction of the ventilation system and the subslab gases. This presentation will focus on the diagnostic procedures which have been utilized and the mitigation steps which have been recommended as a result of conducting the diagnostics. As they relate to diagnostic procedures, geographic differences and building construction details will be discussed.

Key Words : Radon, HVAC, Ventilation, Mitigation

Introduction

The conceptual model for radon entry into buildings has directed field investigations of indoor radon problems towards two areas. These are an investigation of the ways in which the operation of the mechanical equipment in the building affects radon entry and could potentially be used to control radon entry. The second area is the ways in which the foundation and underlying materials affect radon entry and could be used to control entry by soil depressurization.

Conceptual Model of Radon Control and Entry

Both approaches, HVAC and soil depressurization control methods prevent soil air entry by managing the air pressure differential relationships between the air in the soil and the air in the building. In addition dilution by increased ventilation plays a role in the HVAC approach and sometimes plays a role in a soil depressurization approach.

The radon concentration in a school or an individual room of a school is the results of interactions between the climate, occupants,underlying geology, HVAC system and the fabric of the interior partitions and the building shell. In school rooms with radon levels greater than 4 pCi/L the majority of radon enters from the soil or bedrock beneath the building by mass air flow or molecular diffusion through cracks and holes in the foundation. If the building is depressurized relative to the outside air then air pressure differentials draw radon laden soil air into the building. The same pressure difference also draws outdoor air in through the above grade leakage sites, diluting the indoor radon concentration. The net effect on the indoor radon concentration of depressurizing the building depends on whether the increased radon entry overwhelms the effect of the increased amount of outside air entering.

Operation of the HVAC system sometimes has a dramatic effect on both the ventilation rate and the entry rate of soil air. The particular effect depends on the details of the equipment and the triggers it's operation strategy uses. For example, if the system pressurizes the building indoor radon is practically eliminated. However if the building is depressurized it will increase the rate at which radon enters the building.

Radon Levels in Schools

The radon levels in school buildings have been observed to vary considerably spatialy and temporally. Both have been known to vary by an order of magnitude. Figure I shows the room average concentrations of radon for schools investigated. Error bars show one standard deviation to give some

sense of the spatial variation. Temporal variation is often explained by the operation of HVAC equipment. This is discussed at some length in the text.
HVAC Diagnostics

(System Design) In evaluating the expected impact of an HVAC system on measured radon levels, one of the first steps is to review the design of the system as portrayed by the architect/engineer's drawings. In particular it is important to review the planned relationship between exhaust fan rates and outside air supply rates. In addition the planned control sequence and triggering parameters must be considered. The possible effects of various types of system designs have been reported on by Leovic, Craig, and Turner elsewhere.(1)

In general, areas of the building designed for containment of local sources (exhausted areas) might be expected to cause local negative pressures with respect to the subslab, and areas designed to be supplied with filtered fresh outside air might be expected to cause local positive pressures with respect to the subslab.

The resultant overall pressure relationship of the building would be expected to be the combined result of the overall balance of the amount of makeup air vs. exhaust air, the degree of duct leakage, and the tightness of the building shell itself.

Ventilation systems which are designed to keep a building under negative pressure, such as exhaust only systems with leaky window walls would be expected and have been observed to exacerbate radon entry.

(System Operation) Review of system plans is only the first step in evaluating the expected effect of the HVAC system's effect on radon. Equipment observations must be performed in order to determine just what equipment is actually being operated, its operation schedule, the control sequence, and wether the air flows are near the design quantities. Air flow observations may be as simple as visually observing the position of an outside air damper blade or the operation of an exhaust fan and the direction of air flow with a chemical smoke pencil, or may be more involved. Measurement of actual exhaust flow rates with an air balancing hood has proven valuable for determining the operational status of exhaust systems and for conducting a building shell tightness test. Observations of closed outside air intakes or inoperative make-up air supply fans are typical indicators of HVAC system not being operated or maintained as they were design to be.

(Ventilation Air Delivery Rate) In an occupied school with its normal population

of students, the measurement of Carbon Dioxide levels with a portable real time Carbon Dioxide meter has proven very valuable. From these readings inadequate ventilation rates (amount of dilution air) can readily be determined. Current ASHRAE guidelines require a minimum delivery of 7 l/s (15 cfm) of outside air per student in a classroom.(2) Carbon Dioxide readings in classrooms, (taken before lunch time or during mid-afternoon before school lets out), are a good indicator of the ventilation rate occurring on the day of the measurement. Levels over 1000 ppm (with 350 ppm outside) are an indicator of a need for improved ventilation for Indoor Air Quality reasons other than radon dilution.

In a significant number of the schools which have been evaluated in the US EPA SEP program, increasing the observed ventilation rate to meet the current ASHRAE guideline through the use of powered supply make-up air would be expected to lower the radon level to acceptable levels while the system is operating.

(Building Shell Pressure Relationship) A measurement of the pressure relationship between the inside of the building and outside is one of the key parameters which needs to be investigated. This can be accomplished on a not too windy day by simply making measurements through a crack in a closed doorway or window utilizing a sensitive electronic micromanometer (pressure transducer). Readings of slight positive pressure (+.001 to +.010 inches water) will help to keep radon out during operation of the HVAC system, while negative readings will cause increased radon entry.

(Building Shell Tightness Test) In order to determine how much make-up air would be needed to slightly pressurize a building, the equivalent of a fan door pressure test can be performed. The results of this test will reveal the practicality of "building pressurization" to mitigate an observed radon problem.

Foundation Investigation

An investigation is carried out in a school with elevated radon levels that collects information on the foundation and the materials immediately beneath it. The bulk of schools investigated have been slab on grade structures. The material beneath the slab is often sand fill or sand and gravel that was native to the site. A layer of stone pebbles has been found under 1/3 of the buildings investigated to date. This detail provides a plenum like space for lowering the air pressure under the slab with ease.

A vacuum cleaner with a variable speed control is used to assess the airflow resistance of the sub slab material and the distance which a low pressure field can be extended from the suction point. This information can be used to plan the number and location of suction points and the fan performance characteristics of a soil depressurization approach. Suction is ordinarily applied near the outside edge of slabs. This is done because drain tile, stone pebbles or soil settling will most likely be found in this location.

Soil air radon measurements are also made beneath the slab. This has some value in locating "hot spots" and understanding radon sources.

Major limitations in this part of the investigation are finding locations where holes through the slab can be made and repaired unobtrusively. The tests are performed in only one or two areas of the schools, usually in the rooms with the most elevated radon levels. There is the risk that the limited sampling will find areas that are not representative of the school in general or even of the room in which the tests are being made.

Conclusions

A comprehensive approach to Radon diagnostics which includes both HVAC diagnostics and foundation diagnostics has been demonstrated to work effectively. In seven out nine schools investigated an HVAC approach was recommended as the first choice. Often this was the case because the existing ventilation strategy did not meet current ASHRAE guidelines or in some cases state regulation. Many of the HVAC systems in the schools were not operating the way they were originally designed because of operator intervention or disrepair of equipment.

Soil depressurization approaches were considered for each school but only recommended as the first choice in two buildings. These buildings had adequate outside air and could easily control radon by depressurizing under the slab in a small number of rooms.

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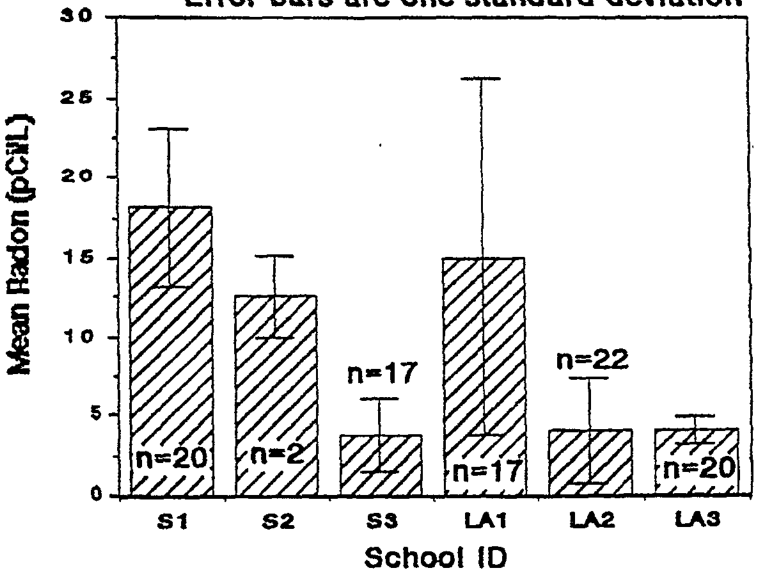
Figure 1 Radon Levels in School Rooms
Error bars are one standard deviation



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BUILDING HVAC/FOUNDATION DIAGNOSTICS FOR RADON MITIGATION IN SCHOOLS: PART 2 TECHNICAL SESSION T-20

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ABSTRACT

Elevated levels of radon have been identified in school buildings throughout the United States and, as a result, the U.S. Environmental Protection Agency's (EPA's) Office of Research and Development has been researching radon reduction techniques for schools since early 1988. Initial research has addressed subslab depressurization, a successful radon reduction technique for many houses.

Radon levels in schools can be influenced by the heating, ventilating, and air-conditioning (HVAC) systems' effect on the pressure relationship of the building envelope. As a result, HVAC strategies for radon control have been used successfully in some schools. This Part 2 presentation focuses on a case study of the diagnostics and mitigation procedures performed in a school researched by the EPA in New York State. Building construction details, radon diagnostic measurements, mitigation system details, and post-mitigation radon levels are discussed.

BACKGROUND

Many scientists believe that children are at a greater risk of developing lung cancer from exposure to radon than adults for a number of reasons: they have smaller lung volumes and higher breathing rates than adults; their rapidly dividing cells may be more susceptible to radiation damage; and they have a longer life-span for latent cancers to develop. In order to reduce the potential health risks of radon exposure to children and those who work in schools, the U.S. Environmental Protection Agency (EPA) has been researching ways of reducing elevated levels of radon in school buildings.

Radon mitigation research in school buildings has shown that heating, ventilating, and air-conditioning (HVAC) systems in schools can dramatically influence the pressure relationship of the building envelope relative to its surroundings and, consequently, affect radon entry(1). If the HVAC system induces negative pressure in the building interior relative to the subslab area, radon entry into the building can be enhanced. Conversely, if the HVAC system pressurizes the building, creating positive pressure in the building relative to the subslab area, it can suppress radon entry as long as this pressure relationship exists. However, school buildings typically have areas such as laboratories, gymnasiums, locker rooms, auditoriums, cafeterias, kitchens, restrooms, and shops that are operated under negative pressure to exhaust chemicals or odors. Even if the classrooms are designed and operated under positive pressure, operation of exhaust fans may cause certain building

zones to be under negative pressure, potentially increasing radon entry in these areas. If pressurization through the HVAC system is under consideration as a long-term solution for radon control, proper operation and maintenance of the HVAC system are critical. Building pressurization with the HVAC system has been effective in reducing radon levels in some schools, depending on system design and operation.

Radon in schools and in houses is frequently reduced by depressurizing the subslab area(2). This technique -- referred to as subslab depressurization (SSD) -- works by inserting pipes through the concrete slab to access the crushed rock or soil beneath. A fan is used to reverse the pressure differential; that is, cause the soil side of the slab to be at lower pressure than the building interior. This reversed pressure relationship prevents radon-containing soil gas from entering the building. SSD systems are commonly more effective when the slab is underlain with a clean, coarse layer of aggregate which helps to extend the field of negative pressure under the slab. Measurements of the pressure fields under a slab are commonly referred to as pressure field extension (or subslab communication) measurements. The reader is referred to Radon Reduction Techniques in Schools -- Interim Technical Guidance for details(3).

If a SSD system is installed in a school to control radon entry, it must overcome any negative pressures that are generated by the HVAC system. Many schools that operate under positive pressure have actually installed SSD systems in order to control radon levels when the HVAC fans are not operating. This paper discusses experience with two approaches for radon reduction in a school researched by the EPA in New York: pressurization with the HVAC system and SSD.

CASE STUDY

This school, located in New York State, is approximately 4450 square meters (sq m) in area. The school foundation is a combination slab-on-grade (3600 sq m) and basement (750 sq m) with a small crawl space (less than 100 sq m). Two-day charcoal canister measurements made in December 1989 while the school was occupied showed that 12 of the 21 classrooms in the slab-on-grade section exceeded EPA's current guideline of 148 becquerels per cubic meter (Bq m^{-3}). The highest screening measurement in the slab-on-grade area was 770 Bq m^{-3}. Measurements in the basement ranged from a low of 230 Bq m^{-3} in the boiler room to a high of 1720 Bq m^{-3} in a storage area. Radon levels in a four-room office area in the basement averaged 1080 Bq m^{-3}. The crawl space, with a poured concrete floor, does not seem to be a major contributor to the school's radon problem.

Description of HVAC Systems

The HVAC system in the slab-on-grade section classrooms consists of individual room unit ventilators mounted on the outside wall in 19 of the classrooms and overhead in the other 2. The unit ventilators are rated at 470 liters per second (l/s), and the minimum and maximum capacities for outdoor air for these units are estimated to be about 80 and 140 l/s, respectively. Note that the New York State Education Department requires that classrooms be supplied 4.7 l/s of outdoor air per student only when the outdoor temperature is above 2°C. Consequently, outdoor air is typically not delivered to the classrooms during much of the winter season. (As a comparison, the most recent ASHRAE ventilation standard (4), 62-1989, recommends that 7 l/s of outdoor air per person be supplied to classrooms.) The HVAC system in this school also includes a number of exhaust fans in selected areas (e.g., corridors, kitchens) that cause the building to be under negative pressure when they are operating.

The HVAC system in the basement area is supplied by an air handling system that *services* only that area. Outdoor air intakes for this system have been disabled; however, some leakage of fresh air into the air handling system is evident. The amount of outdoor air leaking into the system has not yet been quantified.

Boiler room combustion air is supplied at a rate of about 1100 l/s with the boiler operating at low speed. With the boiler operating at high speed, no appreciable difference in makeup air volume was measured. This implies that the passive delivery system is undersized for the boilers when they are operating at high speed, causing additional depressurization of the basement area. The following sections summarize the radon diagnostic measurements performed in this school during the winter of 1989-1990 and subsequent installation of a SSD system in the basement. The slab-on-grade and basement areas are discussed separately.

Slab-on-Grade

To evaluate the potential effectiveness of a SSD system in the slab-on-grade area, subslab communication was measured in two classrooms with elevated radon levels located at opposite ends of the building. Since the slab was poured directly onto a mixture of tightly packed sand, silt, and clay, subslab communication was relatively poor and did not extend much beyond 4 meters (m). Consequently, mitigation by SSD would require many suction points since there are 12 slab-on-grade classrooms with elevated radon levels.

To investigate the potential for radon control by pressurization through operation of the unit ventilators, the outdoor air intakes for seven classrooms in one wing were opened to provide approximately 80 l/s of outdoor air during the occupied periods. (The HVAC system operates from about 7 a.m. to 1 p.m. and is on setback -- with no outdoor air -- during other times.) Spot differential pressure measurements under these conditions showed that the building interior was at a negative pressure of roughly 8 pascals (Pa) relative to the outdoors, even when the outdoor air intakes to the unit ventilators in one wing were opened to their minimum position. This indicates that the negative pressures induced by the exhaust fans and natural stack effect overwhelmed any pressurization by the unit ventilators.

It was not possible to evaluate the effect of the additional outdoor air on radon levels in the classrooms since the outdoor air was not adequately heated by the unit ventilators and, consequently, the rooms were uncomfortably cold. Current research is addressing additional mitigation options in the slab-on-grade section.

Basement

Subslab communication measurements in the basement showed that the subslab pressure field extended about 10 to 12 m from the suction point, indicating that a multi-point SSD system would likely control radon levels in the basement. Subslab communication in the basement seems to be much better than in the slab-on-grade section since the basement slab is underlain by a mixture of sand, silt, clay, and gravel (with the gravel facilitating subslab air flow). Continuous radon measurements collected in the basement office area during December 1989 averaged 1265 Bq m^{-3}. Radon concentrations immediately prior to SSD system installation are not available due to equipment failure.

A five-point SSD system was installed in January 1990. The system ran passively for approximately 5 days. Continuous radon concentrations in the office averaged 773 Bq m^{-3}, a reduction of about 40 percent from the continuous measurements collected in December 1989. On January 31, 1990, the SSD system was activated with a fan (rated at

190 1/s at 0 Pa). Continuous office radon concentrations quickly fell to an average of 414 Bq m^{-3}, a reduction of about 65 percent relative to the December levels and about 45 percent relative to the passive mode.

These initial radon measurements were made prior to excavation of pits at the suction points. Excavation of suction pits, although sometimes difficult depending on the subslab soil type and compaction, is often done to increase pressure field extension. An effort was made in this project to evaluate the effects of suction pit excavation on both pressure field extension and radon levels in the basement.

In early March 1990, suction pits were excavated at each suction point in the system to increase the negative pressure field beneath the slab. At this time, adjustments were also made to ball valves that had been installed in each of the five suction pipes in order to investigate the effects of restricting the flow through a given suction point. Following excavation of the pits and adjustment of the valves, negative pressures were measured beneath the slab in several areas where positive pressure was measured prior to digging the pits and adjusting the valves. Short-term (1-day) radon measurements in the office averaged 296 Bq m^{-3}, showing a reduction of 30 percent from levels prior to pit excavation and valve adjustment, and a reduction of about 75 percent from premitigation levels.

Figure 1 shows the differential pressure (in the subslab relative to building interior) versus distance from the suction point, both before and after the suction pit excavation. The valves were closed at the other four suction points so that P1 was the only point depressurizing the subslab. These data show that excavation of the suction pit decreased the pressures in the test holes close to the suction point and increased the strength and extent of pressure field at the distant holes. In fact, the farthest hole showed a slight positive pressure prior to excavation of the suction pit.

Figure 2 shows the pressure in the suction points under the following conditions: (1) all valves completely open and no suction pits, (2) all valves open and suction pits at all points, and (3) valves adjusted and suction pits at all points. Subslab differential pressure measurements with a micromanometer indicated that two of the suction points were taking advantage of foundation settling to increase the pressure field. Restriction of the valves at those two points helped to increase the pressure-field extension at the other three suction points. This resulted in a more optimal system design despite reduced static pressures at the suction points, as shown in Figure 2.

Additional steps to further decrease radon concentrations in the basement include supplying more combustion makeup air to the boilers to decrease the negative pressure being induced on the basement and introducing outdoor air into the air handling system. It is expected that these additional actions will decrease the basement radon concentrations below 148 Bq m^{-3}. The effects of these additional steps will be evaluated and presented at the conference.

CONCLUSIONS

The following conclusions may be drawn from this school in New York State currently being researched by the EPA:

1. If adequate makeup air is not provided in a school building, negative pressures caused by the HVAC system will enhance radon entry.

2. Although increasing outdoor air supply has been shown to reduce radon levels in some schools by pressurizing the building, this approach does not seem to be a viable year-round option with the unit ventilators in this school because of comfort.

3. Adequate combustion air must be supplied to the boiler room in order to reduce depressurization in the basement area.

4. Subslab pressure field extension can be improved by excavating suction pits and balancing flow through selected suction points.

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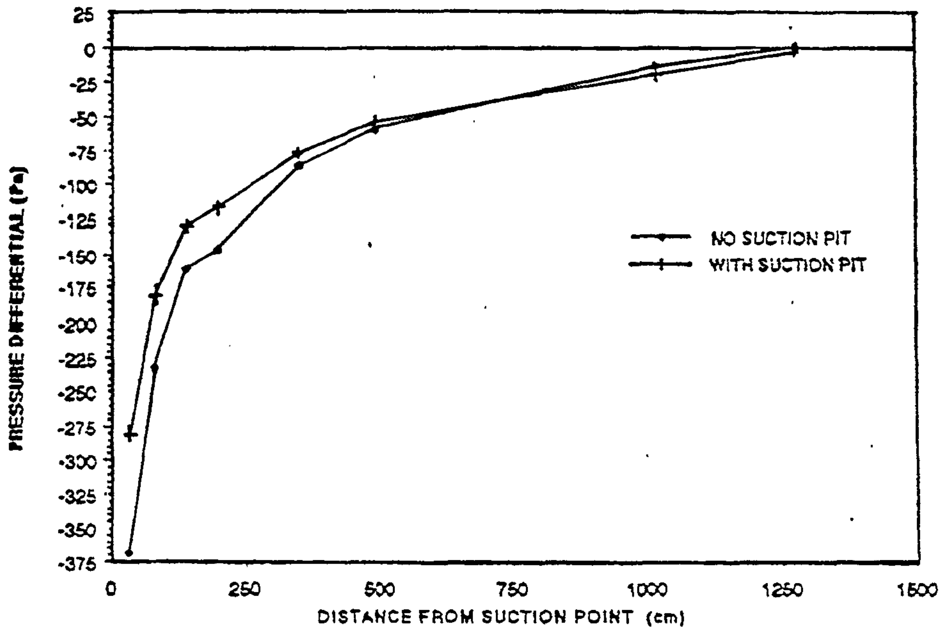

Figure 1. Pressure Differential Versus Distance From Suction Point.

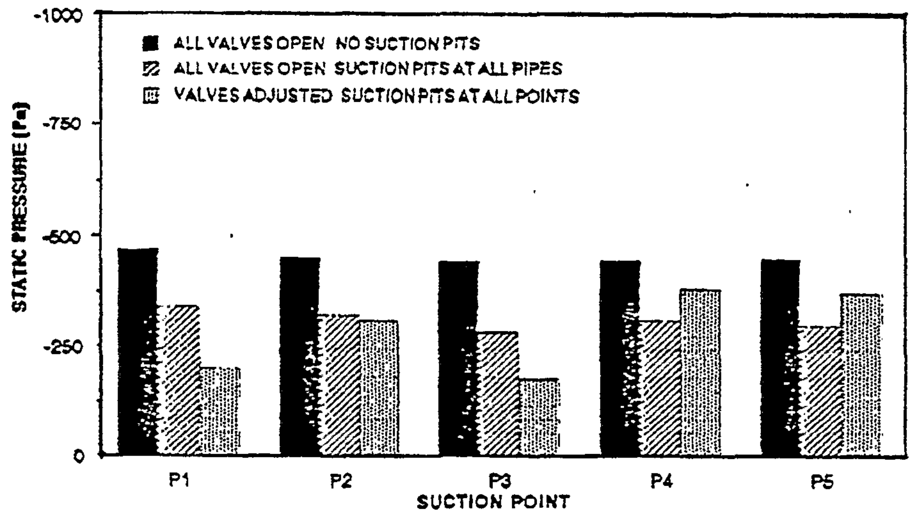

Figure 2. Suction Point Static Pressures.

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LEVIN

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Prevention and Control of Indoor Air Quality Problems

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Critical Building Design Factors for Indoor Air Quality and Climate: Current Status and Predicted Trends

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Abstract

In recent years, some building design professionals have become more aware of the indoor air quality concerns of owners and occupants and as a result, they have made some important changes to improve indoor air quality and climate. These changes include improvements in site planning and design; overall building design; ventilation and climate control systems; and materials selection and specifications. In addition, changes that limit the chemical contamination of building air during the construction process and during occupancy of buildings are also occurring; some of these changes are specified or controlled by design professionals. However, the majority of design professionals have little or no awareness of indoor air quality considerations. There is inadequate dissemination of building science research results to design professionals. There is a need for a useful general body of knowledge, theory, and practice regarding building-environment-occupant interactions. The lack of such knowledge, theory, and practice is an impediment to developing the necessary professional design tools and practices to address effectively indoor environmental quality and energy conservation issues.

KEY WORDS:
Design, Architecture, Site planning, Materials selection, Building ecology

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Introduction

Important changes in building design have taken place in recent years and many of these changes can improve indoor air quality (IAQ) and climate significantly. These changes are being made in response to heightened awareness and concerns of occupants, building owners, and the design professionals themselves. Additionally, building products and materials manufacturers are making and marketing products to improve IAQ (Levin, 1989, 1990a, 1990b).

The changes tend to fall into four major categories: site planning and design; overall architectural design; ventilation and climate control; and materials selection and specifications. Additional design and building professional responsibilities related to the commissioning of newly constructed or renovated facilities have developed. Table 1 lists the major phases and subcategories of critical building design control.

Major IAQ Control Measures by Design

Many design approaches to IAQ control have been used in various projects. In this section we identify some of the dominant trends and approaches to provide an overview of the field. Not all of the IAQ control methods described in this paper are used by even the most air quality conscious designers. Some of them are no more than pro-

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Table 1 Major phases and subcategories of critical building design control

Site planning and design
Ambient air quality
Local source control
Overall architectural design
Vehicle access
Building openings
Operable windows
Pollutant-generating activities
Mix of users in building
Envelope and structural materials
Penetration of volumes
Basement dehumidification
Smoking lounges
Ventilation and climate control
Dilution by outdoor air
Air intakes
Exhaust locations
Air cleaning
Space air distribution
Heat recovery
Microbial control
Standards development
Materials selection and specifications
Low-emitting materials
Preventive installation procedures
In-place curing
Construction process and initial occupancy
Design documentation and commissioning
Special ventilation
Initial occupancy period

posals and have not, to our knowledge, been used at all, although each has been developed within the context of some actual building design process. We have made an effort to identify the nature and extent of use of most of the design IAQ control measures described.

Site Planning and Design

During site planning and design, a few design professionals address sources of indoor air contaminants in ambient air. These sources may originate at a distance from the site or locally. They include gaseous and particulate contaminants generated by motor vehicle, power generation, and industrial process combustion; particulate matter from agriculture, road dust, and wind-generated soil erosion; and ozone formed by the combination of NO_2 and hydrocarbons in the presence of strong sunlight. Soil gas contaminants including radon and organic chemical compounds can enter buildings through gaps in construction elements in contact with the soil or by direct migration through semi-permeable building materials. Table 2 lists the major elements of IAQ control during site planning and design.

There are several control measures that building designers use to control indoor air quality by site planning and design. They evaluate sites prior to acquisition and project planning to avoid problem sites. They locate

Table 2 The major elements of IAQ control during site planning and design

Pre-design site evaluation
Analyse regional and local ambient air quality data
Analyse adjacent and nearby pollutant sources
Vehicular traffic
Industrial sources
Commercial sources
Agricultural sources
Analyse soil and groundwater sources
Radon and other radioactive decay products
Volatile and semi-volatile organic compounds
Determine prevailing weather and wind patterns
Diurnal variations
Seasonal variations
Microclimate
Site planning
Site selection for suitability
Building location and orientation
Vehicular circulation
Local source control
Landscape and architectural buffers
Soil depressurization
Drainage
Site preparation and imported soils

the building and appurtenances on a site distant from pollution sources or pollutant plumes. They plan vehicular circulation and parking to minimize pollutant concentrations at the building edge, especially air intakes and other openings (Levin, 1988, 1989).

Designers and project planners can review and evaluate ambient air quality data and local contaminant sources during feasibility studies. Data are obtained on air quality and climate from local agencies. Soil and water samples are analysed for potential sources of contaminants. Activities in adjacent and other nearby structures are identified to determine potential pollutant emissions. If necessary, air monitoring is performed to evaluate outdoor air quality and proximate pollutant sources. Soils and gaseous soil emissions are tested for volatile organic chemicals (VOC) and radon. Where warranted, mitigation measures including pressure relief systems and more effective sealing of pathways into the building are designed to reduce soil gas or groundwater penetration into the structure (Levin, 1988).

Overall Architectural Designs

The overall architectural concept (or "schematic" building design) includes basic decisions about building shape and size, orientation, layout of floor plans, location of pollutant-generating activities, envelope and interior materials, fenestration, and general ventilation. Experience shows that many IAQ problems result from decisions made at this stage of the project. Although problems resulting from such decisions can be mitigated by subsequent control measures, it is often more cost-effective to consider good IAQ from the outset and to design for it already at the schematic design phase. Very few designers have considered IAQ in the development of the design concept, although the number is growing. Table 3 lists the major categories of overall architectural design considerations.

Table 3 The major categories of overall architectural design considerations

Location of vehicle access	Separate from air entry points
Vehicles in buildings	Provide air supply and exhaust removal, negative pressure to building
Building openings facing clean air	Consider sources, wind, building pressure
Operable windows for backup ventilation	Occupant-controlled for comfort
Isolate pollutant-generating activities	Separate rooms, negative pressure, no recirculation
Durable envelope and structural materials	Minimize emissions, maintenance, refinishing
Basement dehumidification, pressurization	Prevent microbial contamination, pests, soil gas entry
Separate smoking lounges	Exclude polluting behaviour from general space

Building Openings

Openings through which contaminants might enter should be located distant and not downwind from identified pollutant sources outside the building. Interior building areas adjacent to such potential sources of exhaust fumes are positively pressurized to reduce possible entrainment of exhaust. Relationships of spaces with adjacent functions must also be considered.

Operable Windows

In a reversal of the trend toward sealed windows that dominated building designs from the 1960s well into the 1980s, buildings' designed recently have often included operable windows. In some instances these are provided as an "emergency ventilation" system in the event of inadequate ventilation by mechanical means. In many such instances occu-

pants are not actually permitted to open the windows; in some cases they are locked in the closed position. When mechanical ventilation is operated to create positive pressure inside a building, opening windows might not provide additional outdoor air.

Some designers provide operable windows to allow occupants the psychological benefits of direct visual access to the outdoor environment as well as to actually allow air to enter occupied areas directly. The installation of a sensor "interlock" for window operation and the ventilation system controls can maintain a "balance" in the ventilation system while permitting occupant operation of windows. The sensor sends a signal to the ventilation system controls which then compensate for the change in pressure. Such a system has been proposed for the new United States Environmental Protection Agency headquarters facility (Levin, 1988).

Pollutant-Generating Activities

Activities such as food preparation, printing, art, tobacco smoking, certain health care, and others can be sources of contaminants inside buildings. These activities are identified and located away from sensitive areas or occupants. Airflow is controlled to avoid transferring contaminants to adjacent spaces or into a recirculating mechanical ventilation system. Direct exhaust without recirculation is the preferred solution. These IAQ control design measures are being used more frequently and by growing numbers of designers, although consideration is still insufficient in the vast majority of projects.

Envelope and Structural Materials

Materials are chosen that are known to have low pollutant emission characteristics. When such materials are not suitable, alternative means of controlling the contamination of air in the completed building are used including temporary ventilation, in-place curing, and encapsulation or isolation of materials from the building occupants' air. Where glazing permits direct sunlight to strike interior materials, darkened glass or shading devices are used to minimize such sun entry. This reduces surficial heating of materials and associated episodic elevation of VOC emissions. Increasing numbers of designers are concerned about emissions from building materials, but relatively little has actually been done to consider material emissions in most projects.

Vehicle Access

Motor vehicle access to garages, loading docks, and pedestrian drop-off points are located away from air intakes and building entries when designers consider indoor air quality. Where openings do occur near vehicle access points, positive building pressure is maintained inside the opening to keep exhaust fumes out. Spatial relationships of vehicles and occupied building areas must also be considered. This addresses one of the most widely recognized sources of IAQ problems, and growing numbers of designers are considering vehicle access in relation to building openings.

Basement Dehumidification

Control of microbial contamination in basements is accomplished by reducing humidity. Some designers have developed and tested full-scale installations of sophisticated systems to control humidity as well as soil gas entry into basements in Canadian houses. The Canadian system has also been designed to reduce water leakage and cold floors (Walkinshaw, 1990). Further research and development activities are likely to produce refinements and alternative systems to improve IAQ and climate in basements.

Special Provisions for Polluting Activities

During overall building design, identification of occupant activities likely to be sources of indoor air pollutants allows planning for their containment and control. Printing, cooking, art and hobby activities,

and other common building occupant functions can be addressed by locating them where their emissions will not adversely affect other parts of the building. By providing separate, dedicated, and properly ventilated spaces, their impacts can be minimized.

Other activities such as photocopying, food preparation, and graphic arts have also been provided with increased ventilation, no recirculation, and additional exhaust systems to minimize exposure of building occupants to the contaminants they emit. Special exhausts with inlets close to the pollutant source have been proposed to control indoor air contaminant levels effectively (Levin, 1988). Dedicated exhaust systems and spaces with direct exhaust ventilation impact the flexibility of interior space planning resulting in an expanded definition of building core. Considering the control of emissions from polluting activities is becoming increasingly common in building design and is, to some degree, practised by most design professionals. Improved information on sources and their control will enable more adequate consideration.

Smoking Lounges

Public awareness and new laws have resulted in the design of separate spaces for smoking in some public access buildings. One-pass ventilation with no recirculation is usually provided to eliminate exposure of non-smoking occupants to environmental tobacco smoke. There is a strong trend in the United States to limit smoking to designated areas or to prohibit it within the building. Nevertheless, there are still many buildings where the issue remains unaddressed by the designers.

Ventilation and Climate Control

Ventilation is viewed by many as an essential design strategy for IAQ control. They argue, that there are too many sources, the sources are too diverse, and they change over time making it impossible to avoid them by design. In particular, contaminants emitted from occupant activities, personal hygiene products, clothing, and other sources are outside the control of the design professional or builder. Therefore, ventilation including dilution of contaminants with cleaner air (either outdoor air or filtered, recirculated air) is used to control contaminant concentrations to acceptable levels. Table 4 lists the major ventilation design considerations for good IAQ.

Questions exist as to the suitability of outdoor air for such dilution, about the proper approach for strong contaminant sources, about contamination from the ventilation system itself, and about distribution of ventilation air within the building. Filter and air cleaner manufacturers are actively involved at present in developing effective air cleaning methods to remove gaseous contaminants. Virtually no designers thoroughly evaluate outdoor air quality and devise means to control contaminants when required to provide acceptable air quality for supply air used for ventilation. ASHRAE Standard 62 (both 1981 and 1989) requires consideration of the quality of outdoor air used for ventilation, and widespread adoption of the standard by code-promulgating authorities is likely to stimulate attention to this requirement.

Dilution by Outdoor Air

Dilution and removal of contaminants by ventilation continues to be the principal approach to indoor air quality control. The quantity of outdoor air used for this purpose is the subject of much investigation and is a driving force in ventilation system design. The issuance of the revised ASHRAE ventilation standard (Standard 62-1989, "Ventilation for Acceptable Indoor Air Quality") has resulted in some changes, mostly increases, in the quantity of outdoor air required for the ventilation of various types of occupancy (ASHRAE, 1989a). This is the primary, if not the only means of controlling indoor air quality in the majority of building designs.

Table 4 The major ventilation design considerations for good IAQ

Dilution by outdoor air ventilation
- Outdoor air per occupant (OA CFM/p)
- Outdoor air exchanges per hour (OA ACH/h)
- Ventilation based on contaminant source strengths
- Direct exhaust from polluting activity spaces

Air intake locations, design
- Avoid plumes from known and suspected sources
- Avoid standing water and cooling tower drift
- Prevent bird roosting or animal entry at intakes

Building exhaust locations
- Avoid contamination of ventilation air intake by re-entrained exhaust
- Increase height and distance from air intakes

Air cleaning and filtration
- Outside air meets ambient air quality standards:
  - particles: media, electronic filters
  - gases: chemisorption, scrubbers
- Recirculated air meets guidelines/standards

Space air distribution
- Prevent/eliminate "dead zones" and stratification
- Deliver ventilation to occupant breathing zone
- Maintain effective pressurization control
- Balanced supply and return air systems

Heat recovery
- Energy-conserving outdoor air ventilation
- Transfer air for high ventilation rate areas
- Use heat recovery devices where practicable

Microbial control
- Avoid fleecy materials in air stream including ductwork
- Eliminate standing water in drip pans

Even so, delivery of the required quantity of outdoor air to the occupants' breathing zone as required by the ASHRAE standard has not been addressed explicitly in the majority of projects to date.

Ventilation standards based on outdoor air supply per occupant have been shown to be insufficient to control unusual or strong contaminant sources. Adequate ventilation rates must be based not only on the human occupant density but also on the activities that will take place, the types and strengths of contaminant sources, the ventilation system distribution scheme, and the volumetric dimension of the spaces in the building. These are explicit requirements of the Standard 62-1989, but their application has been vigorous in only a small number of building designs to date. It is often difficult to identify most or all of the sources during the early stages of design. A review at the time the actual occupancy is clearly defined might be necessary in order to implement the requirement.

Air Intakes

The known sources of potential contaminants for outdoor air intakes include exhausts from other buildings, motor vehicles, industrial and agricultural processes, and exhausts from the building itself. Many building designers have begun locating building air intakes distant from various building systems including plumbing stacks, kitchen and toilet exhaust air vents, and the ventilation system itself. In large buildings, cooling towers are located distant from air intakes to avoid entrainment of drift containing water treatment chemicals or microbial contaminants.

Exhaust Locations

The location of the building system exhaust is related to that of the air intake. Designers reduce potential re-entrainment of exhaust air by locating outlets downwind and distant from potential intake sites. However, the impact of the exhaust location on IAQ is not considered sufficiently in most projects.

Air Cleaning

Where outdoor air is contaminated, designers specify the use of air cleaning and fil-

tration as appropriate. Designers are choosing more efficient media filters or using electronic air cleaners to reduce concentrations of particulate matter in ventilation air. Gaseous contaminants have been largely ignored until now. However, growing consciousness of outdoor air contaminants and their entry into buildings is leading to the investigation and specification of adsorbents for improving ventilation air quality. When indoor air is recirculated with little outdoor air being introduced, air cleaning and filtration are used to remove contaminants from indoor sources. Particles and gases are removed by the means mentioned above. These air cleaning and filtering devices have been used both in central HVAC components and locally (within or near the occupied space).

Space Air Distribution

Considerable differences of opinion exist among researchers and engineers regarding the extent to which ventilation supply air mixes within building spaces. Designers in some areas have begun introducing supply air through the floor, at desk top, and from sidewall diffusers to improve space air distribution. Return air registers are being carefully placed to avoid short-circuiting of supply air to the exhaust system. Supply and return air systems are being carefully balanced to improve space air distribution.

Partial-height partitions in open-space offices can block the airflow, especially under low flow volume and velocity conditions. Some designers raise partition bases above floors to improve the airflow and ventilation space air distribution. Others have used supplemental fans to improve space air distribution. Desktop supply inlets and inlets mounted in raised floors have also been used to improve space air distribution as well as energy efficiency of ventilation systems.

Induction units, fan coil units, and local (personal) fans have been used. "Personal" or individual control of such supplemental units has provided occupants with more access to airflow and comfort.

Heat Recovery

In order to maintain indoor air quality without unnecessary loss of energy efficiency, heat recovery systems are now being used. When designed to remain in service during maintenance, a high level of performance can be achieved (Greim et al., 1990). This technology is not widely used at present, but increasing pressures for improved indoor air quality and energy conservation are likely to stimulate its use.

Microbial Control

Designers have focused considerable attention on reducing potential microbial amplification within ventilation systems. This has been done by selection of materials to minimize absorption of dirt and moisture that provide niches for microbial colonization. Fibrous insulation has been a particular concern due to the large number of instances in which microbial amplification has been observed on its surface in problem buildings.

Control has been achieved by eliminating or reducing the use of exposed fibrous materials for acoustic control, by placing thermal insulation outside of ductwork, and by thoroughly sealing insulation from circulating air. Many designers are now specifying these measures. The ASHRAE Standard 62 calls for attention to potential problems stemming from man-made mineral fibre insulation. Research has shown that as it accumulates dirt, it becomes more hygroscopic, and when wet can easily host microbiological organisms (West, 1989).

There is a need for improved acoustic and thermal insulation materials for use in mechanical systems so that both insulation and air quality goals can be achieved (Levin, 1990b). Insulations exposed to the air stream can be completely covered with mylar or another vapour barrier to minimize the potential for contamination.

Drip pans for cooling coils have been designed for positive drainage to eliminate standing water contributing to microbial growth. Ductwork, mixing chambers, and plenums are being designed for ready inspection and cleaning to control contamination. Humidifiers are now specified using "dry steam" rather than cold water sprays to minimize the likelihood of microbial contamination. While these practices are not yet widespread, increasing numbers of architects, mechanical engineers, equipment manufacturers, and contractors are implementing them.

Standards Development

ASHRAE's recently published revised standard for ventilation and indoor air quality is widely regarded as the most authoritative guide for designers. However, its actual application is almost always limited to use of the prescribed quantities of outdoor air supply during design of building ventilation systems. Other important requirements such as outdoor air quality or the control of unusual pollutant sources are generally ignored by architects and HVAC system designers.

There is no universal agreement regarding the quantities of outdoor air supply required to maintain good indoor air quality. In fact, a very wide range of values for recommended or required outdoor air supply rates exists; the values appear partly to depend on the purpose of the indoor environment and the type of occupants anticipated in it. Thomas Lindvall of Sweden has presented a range of values for outdoor air supply rates from various authorities (Lindvall, 1989). Table 5 lists various recommended and adopted ventilation rates.

Currently there is discussion about revision of the ASHRAE standard and possible integration of thermal comfort criteria into a single, unified indoor environmental design standard. Due to the complexity of the issues, such an integrated standard is not likely to be developed quickly, if at all. Yet building design professionals must resolve the issues even though the standards writers may not be able to do so. It is likely that the ventilation standard will be revised. The revised standard may include more detailed, specific guidance on acceptable contaminant levels and system design requirements, although a scientific basis for guidance on most contaminants is lacking (Grimsrud and Teichman, 1989).

Table 5 Various recommended and adopted ventilation rates (Lindvall, 1989)

(l/s)[a]	Basis or recommending/adopting group and year
>0.3	2% CO_2 (respiration)
>0.5	1% CO_2 (performance)
>1	0.5% CO_2 (TLV)
>3.5	0.15% CO_2 (Pettenkofer Rule, 1858; body odour)
2.5	ASHRAE Standard 62-1981
3.5	Swedish Building Code 1980
4	Nordic Building Regulation Committee 1981
5 - 7	Berglund et al. (body odour)
8	Fanger et al. (body odour)
7.5	ASHRAE Standard 62-1989
5 - 10	Swedish Building Code 1988
10 - 30	Swedish Allergy Committee 1989
10, 20	Nordic Building Regulation Comm., preliminary 1989
16 - 20	Weber et al.; Cain et al. (Tobacco smoke annoyance)
14 - 50	Fanger et al. (total odour)

[a] *1 litre per second ~ 2 cubic feet per minute*

Materials Selection and Specifications

The selection and specification of materials has been the subject of enormous interest, particularly by designers who believe that source control is the most effective strategy for controlling IAQ. Some designers and authors have suggested reducing VOC emissions into indoor air by the careful selection and installation of building materials and furnishings. Some data are available to allow comparison of emissions from various products. However, no comprehensive set of

data exists nor is it likely to in the foreseeable future due to the large number of products and the variations in them over time. Some designers and their consultants have attempted to evaluate the toxic and irritant properties of emissions in order to choose less harmful or irritant chemicals (Levin, 1989; Girman, 1989). Table 6 lists the major steps in conducting such an evaluation.

Material emission testing has been done by a few research institutions and is beginning to be done by product manufacturers as well (Tucker, 1990). Manufacturers are now advertising products as "low-polluting," "non-toxic," "environmentally safe," and other such claims (Levin, 1990a). While such trends are only in their infancy, significant changes are already occurring.

Some designers are requiring the submission of emissions data by manufacturers before specifying or approving the product for use in a building. Included among the projects where such requirements are imposed are several major governmental projects that are "visible" to manufacturers. This has stimulated the development of testing in laboratories and the writing of standards for the conduct of such testing (Levin, 1990a; Tichenor, 1989; ASTM, 1990).

Table 6 Material selection and specification

Specify IAQ concerns in bid documents:
- health, safety, and comfort of occupants
- manufacturer responsibility for review and assurance

Require submittal of product contents and emission tests

Select low-emitting materials and products

Criteria for evaluating emissions data: minimize
- odorants
- irritants
- systemic toxins
- carcinogens, teratogens

Specify minimal required use of adhesives

Minimize use of fleecy (high surface area) materials

Utilize preventive installation procedures
- ventilation during and after installation
- in-place curing: ventilation, bake-out

Authorities have proposed guidelines for maximum emissions for low-emitting materials and products (Seifert, 1990). Some researchers are now planning to evaluate emissions in terms of various biological responses to them (Tucker, 1990). Others have used sensory responses of the anticipated occupants as a screening technique. In one case, the occupants of a home included two chemically sensitive children. After chemical screening, the children judged the products on the basis of brief exposures (Salares, 1990).

The maintenance requirements and projected life-cycle of materials are design considerations with important implications for indoor air quality. Too often these considerations are not adequately addressed during design. Some designers have developed materials and products that consider these factors, with positive effects on indoor air quality. Evaluation of the results indicate that such efforts can be successful in improving indoor air quality (Fredriksen, 1990). Only a few examples of such efforts have been described in the professional and scientific literature.

Problems remain in adequately characterizing emissions from the thousands of available products. Testing is expensive, time-consuming, and not standardized. Interpretation of results is difficult due to lack of knowledge regarding health effects. Tradeoffs between the significance of toxicity and irritation must be determined. Nonetheless, the concern of designers is leading to the development of cleaner products and the elimination of some of the strongest emitters from the market (Levin, 1989; 1990a).

During the coming years it is likely that the marketing of materials will become in-

creasingly competitive while regulatory initiatives increase pressure on reluctant manufacturers. The likelihood is that some of the strong emitters will be replaced by other products. Researchers will increase our understanding of the factors necessary to select "clean," less harmful building products and furnishings.

Controlling Emission Impacts through Ventilation during Installation

Ventilation procedures are specified to reduce the adsorption of VOC emitted from building materials during installation. These procedures involve one-pass, all outdoor air ventilation continuously during installation of strong emitting materials. This reduces the contamination of "fleecy" materials by adsorption of the solvents and other volatile components of adhesives, caulks, sealants, plastics, and other building materials. It is specified that painting and carpeting be done under maximum ventilation conditions. Continuous ventilation during and after installation is required by designers (Levin, 1988; 1989; 1990a).

Construction Process and Initial Occupancy

Design Documentation and Commissioning

Table 7 lists the major steps available to designers to control IAQ during construction and initial occupancy. Designers wishing to control IAQ now document design assumptions thoroughly and provide clear, detailed descriptions of building systems. This documentation is used to evaluate the completed construction during the "commissioning" phase before occupancy. The building is tested against the design criteria to assure its suitability for occupancy (ASHRAE, 1989b). This procedure is rapidly being adopted by a growing number of designers and owners to provide assurance of good IAQ and other building design specifications.

Table 7 Construction and initial occupancy

Prepare design documentation and commissioning plan
- Document design assumptions
- Document systems characteristics, function
- Plan commissioning process

Special ventilation
- During and after installation of strong sources:
  - all outdoor air, continuous ventilation
  - seal return air plenum, ducts

Commissioning process
- Complete operation and evaluation of HVAC system
- Challenge system under full and part loads

Initial occupancy period
- All outdoor air ventilation, extended hours
- Respond to occupant complaints
- Monitor occupant responses to reduced ventilation

Special Ventilation

Designers have begun to specify extra ventilation immediately before and during initial occupancy of newly constructed or renovated buildings. All outdoor air, 24-hours per day ventilation is used to minimize occupant exposure to emissions from new materials and furnishings. Such extra ventilation also reduces the potential for under-ventilation by incompletely balanced HVAC systems, often characteristic of new buildings with poor indoor air quality.

A special procedure known as a "bake-out" has been used in many buildings, and some initial research results have been reported. The procedure involves raising the building temperature for 48 hours or more while maintaining at least minimal ventilation. The elevation of temperature results in more rapid emission of VOC and a correspondingly reduced contaminant load. The research results indicate that the method has potential to reduce contaminant air concentrations, that the process is not a trivial one in terms of technical requirements, and that

there are some significant additional costs associated with its use. It would appear that pre-treatment of materials prior to installation would be a more efficient approach for many materials. However, for adhesives, paints, sealants, and other products applied on location, it may not be possible to reduce emissions adequately in a reasonable time period without the use of a procedure such as the "bake-out" (Girman, 1989).

Initial Occupancy Period

Due to the presence of many newly installed materials and furnishings, the initial occupancy period is often accompanied by the presence of strong emission sources. In order to control the concentrations of indoor air pollutants from these sources, ventilation protocols are modified. Ventilation systems are set to provide maximum outdoor air (up to 100% where possible) during operating hours to reduce airborne concentrations of VOC. Additional hours of operation are provided, sometimes continuously (24 hours/ day) until emission levels have tapered off. This might be from three to six or eight weeks after installation. Buildings are thoroughly flushed before re-occupancy after any period of vacancy and reduction of ventilation – evenings, weekends, holidays (Levin, 1988). Extra ventilation is often used as a solution to air quality problems. Recently building operators have begun to use it as a preventive measure.

Construction during Occupancy

The occurrence of construction during occupancy is common both in newly completed buildings and during renovation, relocation, or adaptive re-use in older buildings. These situations present challenges to designers, constructors, and building management to avoid exposing occupants to fumes and dusts from construction activities when these contaminants are strongly generated and can produce high concentrations. The problem is especially challenging when the construction area cannot be easily isolated from the occupied areas. The contamination migrates either directly or is circulated by the ventilation system.

Care must be taken to identify the sources of contamination and control them. This can be accomplished, at least in part, by providing temporary ventilation in the construction area while thoroughly isolating it from the occupied zone. Management strategies can provide significant reductions in potential occupant exposure by carefully scheduling both construction and occupant activity as well as temporary occupant relocation. Asbestos or lead abatement projects provide examples of the types of barriers, temporary ventilation equipment, and management strategies that can be employed. These measures are likely to be employed more in the future.

Building Science Research Needs

In spite of a few examples of building design that consider indoor air quality, the majority of design professionals have little or no awareness of indoor air quality considerations. Neither codes nor legal requirements have altered sufficiently to create the necessary awareness and changes in practice.

Part of the problem originates from the fact that there is inadequate dissemination of building science research results to design professionals. Ultimately, it is the building design professional's client who decides whether to address indoor air quality, especially where there are no regulatory or financial requirements to do so.

Information Needs

There is a long time lag in the translation of building science research and related fields into building design professional practice. In most instances, the results from building science research are incorporated into codes, standards, and new products. Additionally, research results are adopted by specialized consultants in new and emerging areas of

practice. But economic and institutional factors slow the transfer process (Schon, 1967).

Building design professionals do not normally read technical and scientific publications. Much of their technical information that does not come from consultants is provided by manufacturers and suppliers of building products and materials. Newly qualified professionals provide another source of information when they have acquired new information or learned new methods during their education.

There is a general lack of information available to design professionals regarding indoor air quality. Architects and their consultants who may wish to incorporate low-polluting materials in their designs do not have available to them clear, simple means of obtaining information on products under consideration. They are not trained or qualified to evaluate "healthy" products; they must rely on some sort of industry standards for testing and labelling or rating systems. They need information on the performance of products with respect to durability, life-cycle costs, maintenance requirements, and health effects.

Table 8 Key actors in the building process

Institutional interests
- Codes and standards organizations
- Professional associations
- Industry and trade associations
- Lenders
- Insurers

Designers/builders
- Designers (architects, engineers, interior designers, consultants)
- Product/material manufacturers and distributors
- Builders
  - contractors
  - trades people
  - equipment suppliers

Owners/occupants
- Owners
- Tenants
- Occupants
- Visitors

Building operators
- Building managers
- Building operators
  - janitorial or custodial staff
  - repair personnel
  - maintenance personnel
  - service personnel
- Contract service and supply organizations

Diverse Interests

There is a complex set of actors involved in the process of making and using buildings. Table 8 lists four major groups of individuals with different perspectives, needs, and relationships to the buildings they affect (or that affect them). Controlling energy consumption, indoor air quality, and other environmental factors involves the complex task of coordinating their diverse inputs and resolving differences in their needs.

Need for a Theory of Building Ecology

The absence of a useful general body of knowledge, theory, and practice regarding building-environment-occupant interactions is an impediment to developing the necessary professional design tools and practices to construct and operate low-pollution, energy-conserving buildings. Design professionals and facilities operators are not equipped to analyse buildings as dynamic entities with important impacts on occupants as well as a dependence upon the outdoor environment.

Borrowing from the approach in the biological science of ecology, we have suggested that researchers and practitioners develop a systems approach to understanding building-environment-occupant interactions. We have coined the term "building ecology" to describe this concept or approach to understanding buildings (Levin, 1981). The concept requires application of a systems approach to understanding, designing, and operating environmental control systems in buildings. It also requires consideration of

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the building as a dynamic and complex entity that continually changes in response to external conditions, occupant activities, and operator interventions. Finally, it requires understanding the complex chemical, physical, and biological processes of a building that influence occupant health and well-being.

Conclusion

Much progress has been made toward improving environmental quality and energy conservation through building design and operation. Site planning, architectural design, ventilation, materials selection, and commissioning have all made contributions. Yet there are still numerous technical problems challenging researchers and professionals alike. The next few years are likely to produce further improvements in indoor air quality by design.

An overall approach to building design involving consideration of the interrelationship of the building, the larger environment, and the building occupants in a complex, dynamic system can contribute much to continued improvement of building environments by architects, engineers, and interior designers. This overall approach, called "building ecology," can also provide the foundation for theoretical advances in the building design professions.

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BUILDING MATERIALS AND INDOOR AIR QUALITY

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New building materials, products, and furnishings are known to emit a large number of organic chemicals into indoor air.[3,5,6,13,14,17,19,21,33,34,36] Building occupants' sickness, irritation, and discomforts are often blamed on the presence of such chemicals in indoor air.[21,31] Most of the chemicals of concern are either volatile organic compounds (VOCs) or semi-volatile organic compounds (SVOCs). VOCs have vapor pressures down to 10^{5} or 10^{6} millimeters of mercury (mm Hg); SVOCs have lower vapor pressures. Building designers, owners, operators, occupants, and product manufacturers are increasingly concerned about problems related to indoor air contaminants emitted from building products and furnishings.[14,34]

EFFECTS OF VOLATILE BUILDING COMPOUNDS ON BUILDING OCCUPANTS

The health effects of most VOCs emitted from building materials are not well understood, but many are known or suspected human irritants and carcinogens. In one study, Lars Molhave of Denmark found that 82% of commonly emitted VOCs are known or suspected mucous membrane or eye irritants, and 25% are known or suspected human carcinogens[18] (Table 1). Thus, exposure to these VOCs is implicated in many problem buildings involving indoor air quality complaints. There is also reason for concern about long-term exposure to low levels of many of these compounds.

TABLE 1. Health Effects of 52 Organic Compounds Emitted from 42 Building Materials*

	Mucous Membrane Irritants (%)	Carcinogens (%)	Odorous Compounds[b] (%)
Unknown	2	75	60
None	17		
Known	42	25[a]	30
Suspected	40		

* From Molhave L: Indoor air pollution due to organic gases and vapours of solvents in building materials. Environ Int 8:117 127, 1982, with permission.
[a] Two compounds are suspected human carcinogens.
[b] Average concentration exceeds estimated or known odor threshold.

Control Measures

Much can be done to reduce building occupants' exposures to emissions from building materials and products. Control measures include careful planning, specifications, and selection, modification, and treatment of products, as well as special installation procedures and proper ventilation system operation.

There have been various efforts to characterize (identify and quantify) emissions from building products. Emission rates or source strengths characterized by the testing of building products and materials can be useful when making decisions regarding product selection and use. The information is also useful when prescribing ventilation system operational protocols to maintain acceptable indoor air quality and when assessing complaints associated with indoor air quality problems.

Solutions. Special procedures can prevent or remedy indoor air quality problems that result from material emissions. Many office buildings now operate under special ventilation protocols prior to or shortly after initial occupancy. These include increased ventilation during material installation and initial building occupancy in order to reduce residual airborne VOC concentrations during the early occupancy period.

A procedure known as a "bake-out" has been used to accelerate the emissions of chemicals from newly installed materials, products, and furnishings.[5,6,14,16] In this procedure (described in more detail in the article by Girman in this issue), the building is heated to temperatures above 90° with very low outside air ventilation for more than 24 hours. The larger the period of elevated-temperature and the higher the temperature achieved, the more effective the bake-out. The effect is achieved by the increase in vapor pressure of the chemicals of concern. They are driven out of building materials and into building air. Then, the building is ventilated with 100% outside air for more than 24 hours before normal occupancy.

The bake-out procedure is most effective in reducing emissions from newer materials. However, it has been applied as a remedy in some indoor air quality problem buildings. Caution should be exercised to avoid damage to the building structure or contents during the bake-out as a result of excessive temperature or humidity changes.[5,6]

Limitations. There are several factors that severely limit the use of product selection to protect occupants from toxic or irritating effects of indoor air contaminants. There is not sufficient knowledge regarding the health and irritation

effects of exposure to very low concentrations of most indoor air contaminants. It is hard to interpret the effects of exposure to low levels of a large number of different organic chemical compounds.[20] Contaminants from other sources, especially occupant activities, can be much greater contributors of VOCs in indoor air than product emissions.[9,28]

Products change over time as a result of manufacturing variations, and products change significantly as a result of their normal aging process when in use. Ventilation system operation variables can significantly affect airborne concentrations of and occupant exposure to chemicals emitted from building products.

In spite of these limitations, a careful review of available data concerning emissions and health effects, as well as the judicious use of ventilation, can effectively reduce occupant exposure to toxins and irritants in building materials, products, and furnishings. The quantity and composition of emissions from substantially similar products can vary significantly. Therefore, building design or evaluation of problems in existing buildings should include careful consideration of material emissions as sources of indoor air contaminants.

BUILDING MATERIALS EVALUATION

Evaluating building materials and furnishings will assist in selecting products for new construction, remodelling, renovation, or refurnishing. The process described below has been used effectively in conjunction with the design of several new buildings, ranging from individual residences to extremely large office buildings. The same process and its criteria can be used to assist in the evaluation of sources considered significant in the etiology of health problems associated with occupancy of particular buildings or building types. When particular chemical compounds are identified in indoor air, possible sources may be identified using published information.[10,12,23,25,27,28,37] Appendix A contains a listing of compounds found in indoor air or in emissions tests of building materials.[34]

Elements of the Materials Evaluation Process

The materials evaluation process may be used as a design tool or after the fact to identify likely sources of contaminants when attempting to explain problems in buildings. The process is divided into four major phases as follows:

1. **Identifying Target Products:** Familiarization with the overall building design and general review of products and materials to identify those considered likely to emit toxic or irritating chemicals in the completed building.
2. **Screening Target Products:** Review of suspect products and materials based on printed information from manufacturers and information in the open literature.
3. **Emissions Testing:** Testing of selected materials to determine chemical content, emissions rate, or change in composition due to environmental exposure.
4. **Evaluation and Recommendations:** Review and analysis of results and recommendations for materials selections, modifications, or handling to control indoor air contamination. Negotiation with product manufacturers, suppliers and installers to modify products or their installation and use.

PHASE 1. IDENTIFYING TARGET PRODUCTS

Literally hundreds of separate products are used in most building projects, whether they be simple residences or complex skyscrapers. In order to make the

materials evaluation activity manageable, "target" products are identified based on the potential for occupant exposure to their emissions and the seriousness of such exposure.

Potential occupant exposure is a function of the emission (off-gassing) characteristics and the quantity and nature of the material used in the building. The seriousness of exposure is a function of the toxic or irritating effects of the emitted chemicals and the susceptibility of the exposed population.[20] A preliminary review of the building products and furnishings will result in a "short list" of target products for more detailed review.

To identify target products, it is important to consider the overall building design, the anticipated use of space, and the possible material and product selections. This is followed by a review of the intended use of major materials. In addition to indoor air quality considerations, the criteria for selection of products may include maintenance requirements, cost, expected useful life, acoustic performance, aesthetic effects, and functional performance. The quantities and applications contemplated for each major product are important considerations.

A material's surface characteristics will affect its emissions rates as well as its potential to act as an adsorbent and re-emitter for airborne VOCs. Textiles, fabrics, and insulation materials usually have very large surface areas. Their texture results in an effective surface area many times larger than their plane geometry surface area.

At this point, all questionable products and materials are considered for screening in Phase 2. In general, the list of target materials will include adhesives, paints, caulks, sealants, and insulations, as well as floor coverings, wall coverings, ceiling system, HVAC duct materials, and most furnishings. Table 2 lists many products of concern in offices.

PHASE 2. SCREENING TARGET PRODUCTS

Screening of target products is done by determining (1) their quantity and distribution in the building, (2) their chemical composition, (3) the stability of chemical substances of concern, and (4) the toxic or irritation potential of their major chemical constituents. The result of this screening process is the identification of products and materials for testing or other evaluation.

Quantitative Assessment. Determine the extent of use and use per unit of floor area or building interior volume. On this basis, materials such as floor coverings and ceilings tiles are considered significant due to the large extent of their use—each has virtually 100% coverage (one square foot of material per square foot of floor area). If the ventilation system uses the concealed space above a suspended ceiling as a return air plenum, then both the upper and the lower surfaces of the ceiling tiles are exposed to the circulating indoor air. Thus, ceiling tiles approach 200% (of floor area) coverage.

Office work station "work surfaces" (desktops) usually have between 15 and 35% coverage, depending on occupant density and work station design. In modern work station component systems, this desktop material is often used for shelving in work station closets, which can add an additional 10 to 20% to the coverage ratio. This material is usually exposed on both upper and lower sides and is considered especially significant due to the normally large amount of contact or close proximity between the office workers and the product. The work surface is often a plastic laminate covering a wood and particleboard core. If the laminate does not completely seal the unit, the interior materials are exposed to

TABLE 2. Typical Materials of Concern in Office Buildings*

- **Site work and foundations:**
  - Insecticides and other soil treatments
  - Waterproofing, particularly petroleum derivatives
  - Fertilizers
- **Structure and envelope:**
  - Wood preservatives
  - Concrete sealers, curing agents
  - Caulking
  - Sealants
  - Joint fillers, gaskets
  - Glazing compounds or gaskets
- **Insulations:**
  - Thermal insulation
  - Fire proofing
  - Acoustic insulations
- **Interiors and finishes:**
  - Subfloor:
    - underlayment (particleboard, plywood, chipboard)
  - Flooring systems:
    - flooring or carpet adhesive
    - carpet backing or pad
    - carpet or resilient flooring
  - Partitions:
    - wall coverings
    - adhesives
    - paints, stains, wood preservatives
    - paneling
  - Furnishings:
    - textiles
    - composite wood products (particleboard, plywood, hardboard, chipboard)
  - Ceiling systems:
    - ceiling tiles
    - panels
- **HVAC systems:**
  - Duct insulations:
    - condensate pan insulation
  - Duct sealants
  - Chemicals:
    - cooling tower water treatment
    - boiler water treatment
    - humidifier water treatment

* From Levin H: Indoor Air Quality Update, March 1989. Arlington, MA, Cutter Information Corp, 1989, with permission.

the air stream and emissions are larger. Completely sealed units will have much lower emissions from the materials inside the core.

The coverage of work station interior partitions (normally about half-height on three and one-half sides of each work station) varies with occupant or work station density. It generally approaches or exceeds 200% of the floor area in open office areas where the work stations are used. In denser installations it can exceed 300%. Again, two sides of the product are exposed to the indoor air, and the product is also in close proximity to the office workers. Furthermore, fabric-covered partitions have very high surface area due to the texture. This creates increased surface area for emissions and for adsorption and re-emission.

Chemical Content. During the screening phase, chemical content is assessed from published general information on building products and materials, information obtained from the building's interior designers, or from manufacturers' and suppliers' product literature and data sheets.[12,25,36] These are obtained by requiring all potential vendors to provide Manufacturer's Safety Data Sheets (MSDSs) for all products assembled by them and the names of suppliers of each product not assembled by them. Additionally, they should be required to provide contact information for each of their suppliers and to request the contact individual to cooperate with the design team. These secondary suppliers and manufacturers are then contacted, and additional MSDSs and other information are obtained.

MSDSs are United States Occupational Safety and Health Administration (OSHA) mandated documents listing all hazardous substances contained in the product they cover. MSDSs are generally available for most products of interest, although they vary in quality, and they may lack actual content information due to "trade secrets" exemptions in the reporting requirements. OSHA requires that MSDSs be available to workers for all hazardous substances to which the worker will be exposed. Thus, whether in a factory or at the construction site, each substance used in building materials, products, and furnishings is theoretically covered by an MSDS.

To illustrate, of the large number of chemicals involved in furnishings, approximately 30 chemicals are used to produce the fabric covering interior partitions. The name of the chemical and its function in the manufacturing process or the finished product should be listed by the manufacturer to provide a more complete understanding of the finished product. The fabric is attached to a metal, lumber, or tempered hardboard frame, usually by an adhesive. The panel contains acoustic material, often fibrous glass batting adhered to a hardboard sheet. There are also metallic components used for the exposed frame of the panel and for the adjustable legs that support the panel above the floor.

In general, aspiring vendors are very cooperative in providing the required information. It should be pointed out that the larger the scale of the project, the more likely vendors will be willing to cooperate. Designers working on smaller projects might aggregate the information collection process for several projects or collect the information over a 1-year or 2-year period to develop an attractive market potential that will induce manufacturer cooperation.

Chemical Stability. Stability (chemical emissions) assessments are done by reviewing the vapor pressure and molecular weight data for chemicals of concern, as identified on the MSDSs. Many sources can be used to obtain the data.[21,27,35] A particularly useful source is the Table of Solvent Drying Time in *Industrial Ventilation.*[2]

Additional information on potential emissions into building air is obtained by reviewing emissions test reports and articles in the published literature.[9,11,14-17,28,32-34,36]

Emission factors can vary significantly—up to a factor of 1000—for different brands of similar products.[11,28,33] Therefore, it is important to obtain as much information as possible about the types and quantities of constituents in a given product. Although such a paper evaluation cannot be definitive, it can be useful in selecting potentially acceptable products. It also can be useful in identifying specific compounds to be measured if laboratory testing is performed.

Toxicity Evaluation. Toxicity or irritation potential of constituent compounds is evaluated using standard reference sources.[1,4,7,22,23,27,29,35] For example, Sax lists a "summary of toxicity statement" or rating (THR) for each substance covered.[27] Ratings of "none," "low," "moderate," "high," or "unknown" are given. Routes of entry are given for specified toxic effects. LD_{50} (lethal dose for 50% of experimental animals) values are given for various exposure routes and experimental species. Human irritation potential and target organs or sites are also listed and carcinogenic and mutagenic assessment is reported.

NIOSH's *Registry of Toxic Effects of Chemical Substances, (RTECS)* 1985–1986, Volumes 1–5 plus a Volume 3a and a User's Guide, provide an annotated listing of toxicity and irritation research for tens of thousands of chemical substances.[23] *RTECS* is available in hard copy or on-line through Toxnet (National Library of Medicine), and on CD-ROM from CCInfo Disc. *RTECS* contains a comprehensive list of alternative trade and generic names by which products may be known or marketed, chemical formulas, and cross-references to the Chemical Abstracts Service (CAS) number for each chemical.

EPA is now developing a data base on building materials emission rates. There also exists a large data base developed by NASA for materials used in spacecraft design and operation. Work currently in progress will make both of these data bases more accessible and useful to the interested professional.

From the screening process, determinations are made regarding materials that will require laboratory testing. A combination of high volatility and moderate toxicity would result in further consideration of the substance and the product. A substance of very low volatility and moderate toxicity would be examined in terms of the quantity of the product and the quantity of the substance in that product. No algorithm has been established for this evaluation; a qualitative assessment is the most reasonable approach, given the limited amount of data currently available.

Results. The results of this screening process allow identification of the products most likely to emit significant quantities of irritating or toxic substances. These usually include the carpet system (carpet, pad or backing, and adhesive), office furnishings (work surfaces, shelving, and interior partitions), and the ceiling tiles. Storage systems, adhesives, caulking compounds, paints, sealants, and wood finishes are also materials of concern. Specific products considered or used for these applications may be evaluated by emissions testing.

PHASE 3. EMISSIONS TESTING

Test Methods. Test methods include bulk testing and air sampling in an environmental chamber or from headspace. Headspace testing involves placing the sample in a closed container for a specified period of time, then sampling the air in the "head space" above the sample in the container. Air sampling can also be done in the completed building prior to, during, and after materials installation to develop air quality profiles of the installation.

Chamber tests can be conducted in a very small chamber (usually less than 0.1 m^3) or in a medium-size chamber capable of accommodating full-size samples. Cut samples create problems of distorted ratios between surface area and edges, and cuts through materials can expose materials not normally exposed in the assembled product. Sealing the edges reduces some of these effects. Room-size chambers can also be used, but they are expensive and require larger quantities of materials.

Ratios of materials surface area and weight to chamber volume and wall area should be kept reasonably similar to the ratios found in actual building

situations. Multiple materials tests may also be run to determine "sink" effects, which are the tendency of materials to adsorb airborne substances on their surfaces and re-release them to the air.

Air movement in the chamber should be at air change rates that approximate those found in buildings—between 0.5 and 5 air changes per hour. Humidity should be controlled during the chamber tests. Relative humidity is generally held close to 50% in most chamber tests. Airflow should be controlled within the chamber to assure good mixing and to minimize unusually high velocities at material surfaces. Introduction and removal of air from the chamber is through perforated headers placed diagonally from each other at the bottom and top of opposite chamber side walls.

Material samples are conditioned by placing them in the chamber at a controlled temperature and under forced air circulation for several hours or even days prior to testing. In order to best meet the purpose of the testing, handling of the material should resemble that employed in actual installation of the material in buildings. Products are stored in factory containers until tested. Once opened, they are kept in a normally ventilated room containing typical, new office furnishings until additional testing is conducted. Complete and careful record keeping is essential to accurate interpretation of testing results.

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Emissions Test Data

Several groups have performed emissions testing. Emissions tests have been done by researchers at EPA in Research Triangle Park, Saskatchewan Research Council, Lawrence Berkeley Laboratory, Oak Ridge National Laboratory, Georgia Tech, NASA, and a variety of private organizations.[11,17,19,28,33,34,36] EPA has begun development of a materials emissions data base.[38] Thus far, there is no comprehensive emissions data base, and it is not likely that such a data base will be produced soon. Any data base that is created will necessarily be of limited usefulness. The number of different types and brands of products used indoors is immense, and variations in each product can occur from one manufacturing run to another. Products are modified or replaced by manufacturers from time to time.[37]

The emissions testing that has been done is most useful in providing insights into the emissions process and in giving a general understanding of the wide range of emissions that might occur among products of similar use. It also allows us to better understand how to test products when such testing is performed. ASTM is currently developing a standard guide for the emissions testing in small chambers.[15] Specific test methods can be developed using this guide.

Small Chamber Testing of VOC Emissions

Recently-reported small chamber tests of VOC emissions from various building materials, furnishings, and consumer products are producing important data for the control of VOCs in indoor air. Among such tests were those performed by Bruce Tichenor at EPA's Air and Engineering Research Laboratory, Research Triangle Park, North Carolina.[32,34] The purpose of the tests was to characterize emissions from a variety of sources of indoor air contaminants and to identify the effect of various factors on emission rates. These factors include temperature, relative humidity, air exchange rate, and chamber loading (material area compared to chamber volume).

Materials were screened using headspace testing and gas chromatography—mass spectrometry (GC/MS) analysis to identify the materials' emissions.

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TABLE 3. Organic Compounds Identified Via Gas Chromatography/Mass Spectrometry*

Material Product	Major Organic Compounds Identified
Latex caulk	Methyl ethyl ketone, butyl propionate, 2-butoxyethanol, butanol, benzene, toluene
Floor adhesive (water based)	Nonane, decane, undecane, dimethyloctane, 2-methylnonane, dimethylbenzene
Particleboard	Formaldehyde, acetone, hexanal, propanol, butanone, benzaledhyde, benzene
Moth crystals	Para-dichlorobenzene
Floor wax	Nonane, decane, undecane, dimethyloctane, trimethylcyclohexane, ethylmethylbenzene
Wood stain	Nonane, decane, undecane, methyloctane, dimethylnonane, trimethylbenzene
Latex paint	2-Propanol, butanone, ethylbenzene, propylbenzene, 1,1'-oxybisbutane, butyl propionate, toluene
Furniture polish	Trimethylpentane, dimethylhexane, trimethylhexane, trimethylheptane, ethylbenzene, limonene
Polyurethane floor finish	nonane, decane, undecane, butanone, ethylbenzene, dimethylbenzene
Room freshener	Nonane, decane, undecane, ethylheptane, limonene, substituted aromatics (fragrances)

* From Tichenor B: Organic emission measurements via small chamber testing. In Indoor Air '87: Proceedings of the International Conference on Indoor Air Quality and Climate, Vol. 1. Berlin, Institute for Water, Soil and Air Hygiene, 1987, pp 8-15.

Emissions tests were conducted using a small chamber (166-liter) with carefully controlled temperature, relative humidity, and airflow. The wet materials were placed in the chamber shortly after application to the test surface, and the first sample was collected 30 minutes after the chamber door was closed. Samples were collected on a tenax-charcoal combination and thermally desorbed. Approximately 10 compounds were analyzed by gas chromatography-flame ionization detection (GC/FID).[32]

Several types of materials were evaluated, including silicone caulk, floor adhesive, floor wax, wood stain, moth crystal cakes, and particleboard, among others. Organic compounds identified by the chamber tests are listed in Table 3. Note that several of the compounds on the list affect the central nervous system, some are known irritants, and several are known or suspected animal carcinogens or teratogens.

Table 4 shows the effect of air exchange rate and temperature on the emission rate of moth crystal cakes. As can be seen, the effect of temperature is

TABLE 4. Moth Crystal Emission Factors, Para-dichlorobenzene ($\mu g/cm^2$-hr)

Air Exchange Rate (air changes per hour)	Temperature = 23°C	Temperature = 35°C
0.25	1250	4600
0.5	1400	4850
1.0	1750	5700
2.0	2000	6700

quite large, and the effect of air exchange rate is significant also. The air exchange rates used are representative of the range of rates encountered in residences and public buildings such as offices or schools. The effect of temperature on emissions from moth crystals would be similar to that of other VOC sources.

The effect of time on the emission rates is shown in Figures 1-3.

Implications. Tichenor's work demonstrates that emission rates for "wet" materials can decrease rather sharply during the first few hours after application. However, a critical factor in the rate of decrease is the ventilation rate. For the caulking compound tested, emissions were close to zero after 6 hours at 1.84 ACH. However, at 0.36 ACH, significant emissions were still observed at 10 hours and the emission rate was declining very slowly (see Fig. 1). Emission rates were reasonably similar for three compounds, C_4 ketone, C_8 alcohol, and C_7 ester (see Fig. 2). This supports the notion that maximum available ventilation should be used during and immediately after the application of these materials.

It is noteworthy that concentrations of many of the compounds emitted from floor wax at 0.5 ACH decreased sharply during the first 12 hours from 10^3–10^4 $\mu g/m^3$ to 10^2 $\mu g/m^3$ The concentrations were still at 10^1 $\mu g/m^3$ and decreasing at 48 hours when the experiment was stopped. This suggests that floors that are waxed on a Friday afternoon will still be emitting significant quantities of several compounds on Monday morning and beyond. This is true even with ventilation rates greater than normally encountered during weekends in unoccupied (no mechanical ventilation) schools and office buildings.

Based on the results of chamber, headspace, and test house environments, EPA researchers have constructed a model for predicting indoor air VOC

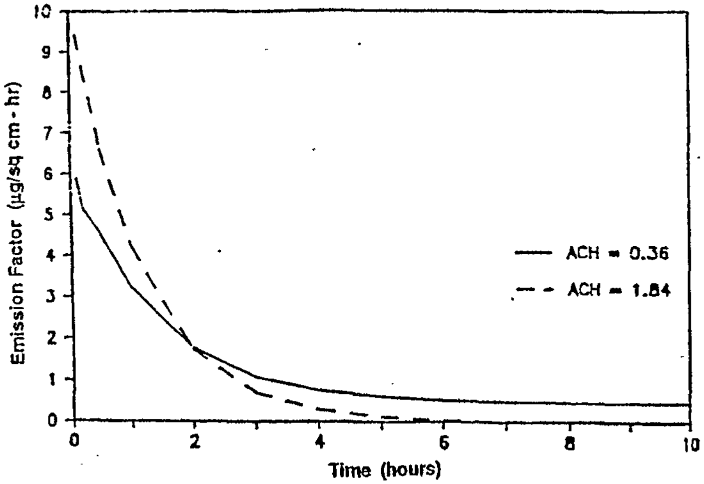

FIGURE 1. Emission factor vs. time—caulking compound. Total measured organics. From Tichenor B: Organic emission measurements via small chamber testing. In Indoor Air '87: Proceedings of the International Conference on Indoor Air Quality and Climate, Vol. 1. Berlin, Institute for Water, Soil and Air Hygiene, 1987, pp 8-15.

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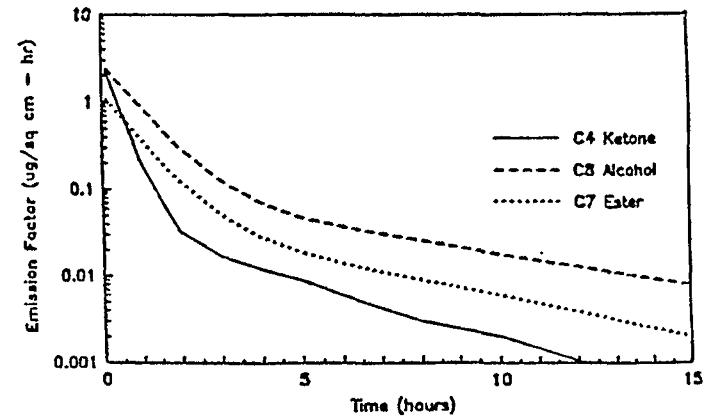

FIGURE 2. Caulk emissions vs. time—three compounds. T = 23°C; RH = 50%; ACH = 0.36. From Tichenor B: Organic emission measurements via small chamber testing. In Indoor Air '87: Proceedings of the International Conference on Indoor Air Quality and Climate, Vol. 1. Berlin, Institute for Water, Soil and Air Hygiene, 1987, pp 8-15.

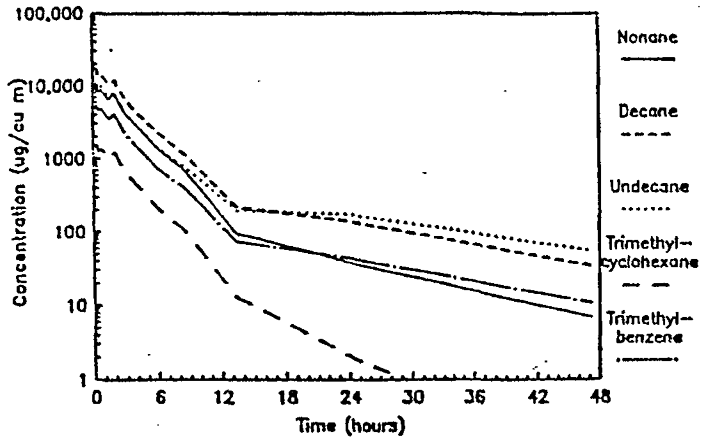

FIGURE 3. Concentration vs. time—floor wax. T = 22°C; RH = 50%; ACH = 0.5. From Tichenor B: Organic emission measurements via small chamber testing. In Indoor Air '87: Proceedings of the International Conference on Indoor Air Quality and Climate, Vol. 1. Berlin, Institute for Water, Soil and Air Hygiene, 1987, pp 8-15.

TABLE 5. Typical Emission Rates for Sources in a 400 m^2 Office Area* (Total Vapor-phase Organic Compounds, Except as Noted)

Source†	Condition	Emission factor (mg/m^2-h)‡	Assumed amount (m^2)	Emission rate (mg/h)
Silicone caulk	<10 hours	13	1	13
Silicone caulk	10-100 hours	<2	1	<2
Floor adhesive	<10 hours	220	30	6600
Floor adhesive	10-100 hours	<5	30	<150
Floor wax	<10 hours	80	100	8000
Floor wax	10-100 hours	<5	100	<500
Wood stain	<10 hours	10	100	1000
Wood stain	10-100 hours	<0.1	100	<10
Polyurethane wood finish	<10 hours	9	100	900
Polyurethane wood finish	10-100 hours	<0.1	10	<10
Floor varnish or lacquer	NA	1	100	100
Particleboard	2 years old	0.2	300	60
Particleboard (HCHO)	New	2	300	600
Plywood paneling (HCHO)	New	1	1000	1000
Chipboard	NA	0.13	300	39
Gypsum board	NA	0.026	1000	26
Wallpaper	NA	0.1	1000	100
Latex-backed carpet (4-PC)	1 week old	0.15	400	60
Latex-backed carpet (4-PC)	2 weeks old	0.08	400	32
Moth Cake (para)	23C	14,000	0.1	1400
Dry-cleaned clothes (perc)	0-1 day	1	6	6
Dry-cleaned clothes (perc)	1-2 days	0.5	6	3

Notes:
Para = paradichlorobenzene
HCHO = formaldehyde
Perc = perchloroethylene (tetrachloroethylene)
4-PC = 4-phenylcyclohexene, an odorous constitutent of some latex-backed carpets
NA = not available

* From Tucker WG: Emissions of air pollutants from indoor materials: An emerging design consideration. Presented at the 5th Canadian Building and Construction Congress, Montreal, Canada, November 27-29, 1988, with permission.

† Emissions data are typical only for the specified brands, models, or units that have been tested; the data do not represent all products of the source type listed. Product-to-product variability can be very high.

‡ Typical values selected by author based on data in "Database of Indoor Air Pollutants" (described in ref. 38).

concentrations.[30] Source strengths and ventilation rates were used in the model to compute some typical values which are shown in Table 5.

Sealants and Caulks

Tests of a variety of sealant products performed by researchers at the Saskatchewan Research Council have found large variations in weight loss, rate of weight loss, and calculated complete drying times[11] (Table 6). A styrene ethylene butylene sealant lost 37% of its original weight during the first 48 hours and was projected to lose 61.7% upon complete drying at 79.3 hours. The emissions were petroleum hydrocarbons and xylene,

TABLE 6. Emissions Data from Various Sealant Products*

Product description	Weight loss in % of original sample @ 48 hrs	@ fully dry	Calculated complete drying time (hrs)	VOC	THR*
Styrene butadiene rubber compound	16.4	35.26	253.7	Aliphatic hydrocarbons Xylene	MOD MOD
Oleoresinous	0.68	4.42	1962.4	Aliphatic hydrocarbons	NA
Polysulphide one-part	0.56	6.2	4931.9	Toluene	MOD
Butyl rubber	5.26	17.69	434.3	Aliphatic hydrocarbons	NA
Acrylic emulsion latex	5.48	11.80		None detected	
Acrylic solvent-based	3.26	13.51	1052.2	Xylene	MOD
Polyvinyl acetate-based emulsion	16.85	26.61		Negligible quantities	
Vinyl-acrylic emulsion latex	12.70	30.05	317.3	Petroleum hydrocarbons	NA
Asphaltic one-part	1.37	8.09	4496.3	Petroleum hydrocarbons	NA
Neoprene one-part	18.00	32.75	214.0	Xylene	MOD
One-part chlorosulfonated polyethylene	4.4	14.38	446.7	Xylene	MOD
Polyurethane one-part	1.2	14.86	8269.4	Xylene	MOD
Silicone	2.06	4.49	487.2	Xylene	MOD
Polybutene	2.39	9.19	627.8	Petroleum hydrocarbons	NA
Styrene butadiene rubber	14.0	19.25	106.3	Xylene	MOD
Neoprene blend	17.4	23.21	101.4	Methyl ethyl ketone Xylene Toluene	MOD MOD MOD
Styrene butadiene	21.1	25.03	55.5	Hexane Toluene	LOW MOD
Styrene ethylene butylene styrene	37.07	61.7	79.3	Petroleum hydrocarbons Xylene	NA MOD
Nitrile	31.5	59.6	271.5	Methyl ethyl ketone	MOD

* From Jennings D, Eyre D, Small M: The safety categorization of sealants according to their volatile emissions. Ottawa, Ministry of Energy, Mines and Resources, Government of Canada, 1988.

* THR = Summary toxicity statement from Irving Sax, Dangerous Properties of Industrial Materials, Fifth Edition. New York: Van Nostrand Reinhold, 1979. MOD = moderate.

A styrene butadiene rubber compound was calculated to lose 35% of the original sample weight when fully dried at 253 hours. Of the original weight 16.4% was lost in the first 48 hours. The emissions were aliphatic hydrocarbons and xylene.

Meanwhile, a one-part chlorosulfonated polyethylene product lost only 4.4% of the original sample weight in the first 48 hours and a calculated 14.4% when fully dried. The emissions were primarily xylene and the complete drying time was estimated at 447 hours.

A one-part polyurethane lost nearly the same amount when fully dried but lost only 1.2% of the original sample weight after 48 hours. Its emissions were also primarily xylene. Its complete drying time was estimated at 8,269 hours, nearly a year,

Silicone caulk lost 2% of its original weight after 48 hours. The researchers calculated a total loss of 2.5% in the next 487 hours. The VOC emissions were xylene, considered moderately toxic by Sax.[27] A summary listing of the Saskatchewan research is contained in Table 6.

From Table 5 it is evident that product characteristics can vary considerably. A compound may have very high emissions but dry rather quickly. Another may have low total emissions and dry slowly. Every other combination is also found. These facts make it clear that it is important to obtain actual performance data on the products, that it does make a difference which products are chosen, and that there is no correlation between total emissions and complete drying time.

Slow-drying compounds or products are the worst from an indoor air quality perspective unless their emissions are either nontoxic and nonirritating or negligible. Fast-drying products like the styrene butadiene rubber compound emit significant fractions ($^1/_3$–$^3/_5$) of their total weight, but they do so in a matter of 3 to 10 days, mostly in the first 2 or 3 days. Thus, by applying these products while using adequate ventilation, the product is reasonably acceptable for indoor air.

The size of the bead is also variable. The tests were done with beads 6 m by 6 mm by 304.8 mm (19.68 ft × .24 in × 1 ft). Bead size affects the emission rate. Emission processes are a function of evaporation from the surface and diffusion through the material to the surface. Of course, a flat section will have more surface area and less interior volume than a round section. Drying or evaporation will be quickest from the surface and slowest from the center. The further vapors must travel to reach the surface, the slower the drying time.

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The physical structure of the material will also affect the outgas rate, although this cannot be predicted precisely without considerable research. Some materials quickly form a skin on the surface. This skin inhibits outgassing after it is formed. However, experimental testing of various products by the Saskatchewan researchers showed that after a few hours, the emissions tend to become more consistent among products regardless of some irregularities during the first few hours.[11]

PHASE 4. ANALYSIS AND RECOMMENDATIONS

The most effective way to obtain "clean" products is to place as much responsibility as possible on the product manufacturer to control emissions and to provide data. A number of standard "guide" specifications have been developed that can be used to direct the potential vendors to provide products and information about them.[15a]

General Considerations

Certain materials are more important sources of VOCs than others. These include carpets, adhesives, caulks, sealants, paints, insulations, and office work station furnishings. Each of these products is addressed below. Suggested approaches for any of these materials may be applicable for other materials, although they are not repeated in detail for each product.

Carpet

Carpet installations are frequently implicated in indoor air pollution incidents. There is not enough information currently available on carpet emissions to permit selection or detailed screening of products on the basis of carpet composition or emissions.

Manufacturers of commercial carpet products are keenly aware of indoor air quality issues, and several have initiated testing programs. Data from these tests can be helpful in evaluating candidate products. Manufacturers may use the data to modify their products or to use in the event of a lawsuit involving their products. Their activities also may result in marketing themes based on claims of low emissions.

Of course, different test procedures will produce different results. If you receive emissions data from a manufacturer, be sure to request a copy of the test method. The history of the sample prior to testing can also significantly affect test results. Important factors include exposure to air movement, temperature, and conditioning prior to testing.

Levels of emissions decline rather rapidly. In some tests, the reported decreases are on the order of 10 to 50 times within the first 3 to 6 weeks of exposure to the environment after installation.[11,14,32,34]

PLANS AND SPECIFICATIONS

Products such as carpets are normally specified in the "construction documents," which consist of "working drawings" and "construction specifications." Specific designations of materials, equipment, and layouts are developed in the preparation of the contract documents. Products are considered and selected. Here architects, engineers, interior designers, and their clients can exert enormous influence on the providers of the products.

Model "Guide" Specifications:

Following are some examples of carpet specifications that can be generally adapted for many building products. Specific language should be developed for each product of concern.

1. Carpets (or whatever product) shall be designed, manufactured, handled, installed, and maintained in a manner that will produce the least harmful effects on occupants of the building.
2. The manufacturer of the carpet shall avoid unnecessary use of chemicals that are toxic or irritating to humans in the manufacture, treatment, or handling of the carpet products.
3. The manufacturer shall implement measures to reduce as much as possible installed carpet chemical emissions that are toxic or irritating to humans.
4. The carpet manufacturer shall provide a specification for the installation of the carpet that uses the least quantity of adhesive necessary to satisfactorily maintain the required performance of the carpet product. Furthermore, adhesive selection shall be based on the lowest level of volatile organic chemical emissions released into the building air. Where more than one adhesive is suitable and the emission rates are similar, the adhesive with the least toxic or irritating contents shall be specified.
5. Manufacturers of carpet shall submit the following for review by the owners and their agents:

- A list of all chemicals used in the manufacture of the carpet. This list shall include a breakdown of the contents by weight, volume, or both.
- A description of any procedures used by the manufacturer to minimize the emissions of VOCs from their product(s).
- A description of all testing performed by the manufacturer, its agents, contractors, or any other party that provides information on the chemical composition of the finished product; the emission rates of VOCs from the

finished product; and a list of all chemicals found in emissions testing, headspace testing, or other tests providing evidence of the emission products, quantities, and rates. For all such tests, a description of the test methods used, the history and conditioning of the samples tested prior to the test process, the raw data obtained from such tests, the agency performing the tests, and the reported results of the testing process should be included.

Submittals Review. The submittals in response to the specifications should be reviewed and compared with any available emission test results for each candidate product. On the basis of the submitted data, including but not limited to the test results, the designer or investigator can determine the need for additional testing or other evaluations.

Conditioning Carpets

The designer or health professional can determine the need for carpet product conditioning prior to installation in the building based on the data obtained from manufacturers and testing. Conditioning can occur at the factory prior to shipment, at the site prior to installation, or *in situ.*

VOC emissions are a function of material temperature, air movement above the carpet, concentration of VOCs in the air above the carpet, and the distribution of VOCs in the carpet. Elevating temperature, maintaining good air movement above the carpet, and providing good ventilation accelerates emissions. Since most floors are relatively massive, it takes far longer to raise their temperatures measurably than it takes to increase air temperature. Trying to condition carpets in place may take several days and perhaps as long as a week to achieve any real effect.

It is far better to condition carpets at the end of the manufacturing process. A conditioning step after manufacturing might involve running the carpet through a well-ventilated, heated chamber. The carpet manufacturing process involves several steps in which the product is heated to very high temperatures, but this takes place in closed chambers. Thus, emissions do not escape, although they may move from the interior to the surface of the product. Chemical changes also occur during the heat cycles.

VOCs may be loaded on the surface of carpet fibers as a result of the process described above. When carpets are first exposed to the air in a building, there is a burst of VOC release; this explains the noticeable odor. If this burst occurs outside the building, this will considerably reduce the amount of off-gassing that will occur indoors.

Whether it is done at the factory, in a warehouse, or outdoors, it is worth considerable extra effort to provide for off-gassing after bringing the carpet product into the building.

Adhesives

Carpet and other flooring adhesives are also sources of indoor air pollutants. Adhesive specifications are typically prepared by the carpet manufacturer. Many indoor air quality concerns may be addressed by minimizing the quantity of adhesive used and the toxic or irritating chemical constituents of the adhesive. Maximizing ventilation during the carpet installation can reduce VOC residues from carpet adhesives.

EPA's Public Access Buildings Study found VOC emissions nine times greater from carpet adhesive than from the carpet for which it was used. Adhesive emissions may occur faster, however, and long-term comparisons were not conducted in that study.[28]

The following can be included in the carpet adhesive specifications:

- Clearly stated concern about indoor air quality and potential chemical emissions from the adhesive product. Specific concerns include odor, irritation, and toxicity of adhesive emissions.
- The adhesive specification by the carpet manufacturer should call for the smallest quantity of adhesive consistent with the requirements of product application.
- The adhesive should have the lowest content by volume of toxic or irritating chemicals while meeting with the requirements for product application.
- The manufacturer(s) of the candidate adhesive product(s) should be required to submit the same type of information suggested above for carpet manufacturers.

The submittals should be reviewed to determine the need for further evaluation, testing, or modifications of the products. If data are obtained for several products, the results can be compared. Investigators should not hesitate to ask manufacturers and suppliers questions. Answers to important questions should be obtained in writing. If the data are difficult to interpret, an industrial or analytical chemist, industrial hygienist, or other qualified professional should be retained to assist in the interpretation.

Temporary Ventilation

Temporary, special ventilation may be required during and immediately following carpet installation to reduce the airborne concentrations of carpet and carpet-adhesive vapors. In general, such ventilation is almost always a good idea. The longer the ventilation period after installation, the lower the residues when the space is occupied.

The contract specifications should require that the HVAC system be operational prior to the installation of the carpet. The preferred HVAC system operation would use supply air fans and ducts only; exhaust ducts would be sealed and exhaust fans disabled. Exhaust would be provided through windows (if operable). This will reduce contamination of return air ducts, plenums, and insulation materials. If operable windows are not present, temporary openings can be created by temporarily removing window glass. In some special cases, temporary exhaust fans will be needed to pull exhaust air from deep interior locations. Stair towers and other paths to the exterior are useful for exhausting air from the building during the temporary ventilation. This temporary ventilation approach is also useful during painting, installation of furnishings, and other such operations both during construction and after occupancy.

Vinyl Composition Tile and Other Flooring Products

Air quality considerations for many other flooring products are similar to those for carpet and carpet adhesive. Minimizing the quantity of adhesive used, the toxic or irritating chemical components of the adhesives, and the emissions from the product are the goals. Specifications should advise contractors and manufacturers about indoor air quality and require submission of

the relevant data. Temporary ventilation will be useful in reducing air levels and residues of emissions.

Caulks, Sealants, Glazing Compounds, Joint Fillers

These "wet" building products may contain and emit VOCs after installation in the building. Considerations similar to those for adhesives apply. The range of measured test emissions is very great. Therefore, selecting products with low emissions and low content of toxic or irritating components can significantly reduce occupant exposure to indoor air pollutants.

Specifications for "wet" building products should follow the same general procedures described above for adhesives. Specifications should require use of the minimum quantities of these materials necessary to perform the required function, adequate ventilation during and after installation, and data from the manufacturers on contents and emissions.

Paints

Paint products contain a variety of VOCs incorporated as drying agents, flattening agents, mildewcides, fungicides, preservatives, and others. These VOCs have been measured in indoor air many months after application of the paints. There is a wide range of formulations with an equally wide range of emission rates and chemical contents. Data from the EPA Public Access Buildings Study showed a hundredfold difference in the VOC emissions from one latex paint and another[28] (see Table 7).

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Specifications should be similar to those discussed for the products identified above. Candidate paint products should be evaluated according to procedures described above for adhesives and other "wet" products. Maximum feasible all-outside-air ventilation should be used during the application of paints to accelerate emissions and remove residues from the building.

Insulations

Insulation materials emit indoor air contaminants from their original composition. They also re-emit chemicals that are absorbed on their very large surface areas. Insulations used for acoustic control are often "fleecy" in order to enhance their sound absorption capabilities. However, the fleeciness enhances adsorption of VOCs and retention of VOCs within the building.

Acoustic insulations, especially those used in HVAC duct work, are particularly challenging from an indoor air quality perspective. Fleecy duct linings inevitably become contaminated by particles and by biological aerosols, which leads to microbial amplification. Covering them with impermeable membranes reduces their effectiveness for noise-control purposes.

It is most important to limit acoustical insulation application to essential uses. Where acceptable, apply it to the exterior of ductwork. Use sound baffles rather than insulations where they will do the job.

Thermal- and fireproof-insulation materials do not necessarily need fleecy surfaces in order to work. However, the economical manufacture or application of the materials often results in a fleecy surface. Where possible, they should be coated with a smooth and impermeable membrane to reduce the adsorption of VOCs on their considerable surfaces.

To reduce the potential for microbial growth on acoustic and thermal insulations, keep fibrous duct insulations clean and limit humidity within the

TABLE 7. Summary of Emission Results

	Emission Rate (µg/m²h)			
Sample[a]	Aliphatic and Oxygenated Aliphatic Hydrocarbons	Aromatic Hydrocarbons	Halogenated Hydrocarbons	All Target Compounds
Cove adhesive	a	a	a	5,000
Latex caulk	252	380	5.2	637
Latex paint (Glidden)	111	52	86	249
Carpet adhesive	136	98	—[b]	234
Black rubber molding	24	78	0.88	103
Small diameter telephone cable	33	26	1.4	60
Vinyl cove molding	31	14	0.62	46
Linoleum tile	6.0	35	4.0	45
Large diameter telephone cable	14	20	4.3	38
Carpet	27	9.4	—	36
Vinyl edge molding	18	12	0.41	30
Particleboard	27	1.1	0.14	28
Polystyrene foam insulation	0.19	20	1.4	22
Tar paper	3.2	3.1	—	6.3
Primer/adhesive	3.6	2.5	—	6.1
Latex paint (Bruning)	—	3.2	—	3.2
Water repellent mineral board	1.1	0.43	—	1.5
Cement block	—	0.39	0.15	0.54
PVC pipe	—	0.53	—	0.53
Duct insulation	0.13	0.15	—	0.28
Treated metal roofing	—	0.19	0.06	0.25
Urethane sealing	—	0.13	—	0.13
Fiberglass insulation	—	0.08	—	0.80
Exterior mineral board	—	0.03	—	0.03
Interior mineral board	—	—	—	—
Ceiling tile	—	—	—	—
Red clay brick	—	—	—	—
Plastic laminate	—	—	—	—
Plastic outlet cover	—	—	—	—
Joint compound	—	—	—	—
Linoleum tile cement	—	—	—	—

[a] Emission rate for cove adhesive is a minimum value; sample was overloaded. It is estimated that cove adhesive is one of the highest emitters of volatile organics.
[b] No detectable emissions.

ducts. Prevent condensation by properly locating humidification or dehumidification equipment and by minimizing condensate "blow-off."

Be sure that lined ductwork and plenums can be easily inspected and cleaned.

Work Station Panels

Because of the very large surface area of work station panels, they are an extremely important factor in indoor air quality. Their VOC contents can be

emitted to indoor air. Their fabric covering can serve as an adsorption surface for VOCs emitted from other products or occupant activities and then act as a "secondary source" of emissions.

One type of interior partition (work station panel) contains chipboard material used for septums. While only 1/16 inch thick, it is made from recycled paper materials similar to the composition of food boxes or the chipboard found in tablets of writing paper.

If inspectors find the panel fabric is soiled, it is cleaned in the plant before being bagged in polyethylene film for shipment. About 25% of panels must have some cleaning. Cleaning is with 1,1,1,-trichloroethane (TCA; methyl chloroform), a common cleaning solvent. It is a relatively volatile compound, which means that most of it will evaporate rather quickly when exposed to air in a well-ventilated space. It also means that a substantial portion of the residue on the fabric at time of packaging will be released in the building when the packaging is removed.

TCA is one of many known eye and mucous membrane irritants commonly found in indoor air. It is also used as a pesticide and in textile processing. Because of its irritant potential, its concentration in indoor air should be minimized through all reasonable measures.

If possible, allow TCA-cleaned panels to be aired out before being packaged for shipping. The furnishings industry is becoming much more aware of indoor air quality concerns, and some manufacturers are quite willing to discuss measures to minimize problems.

One large manufacturer of office furnishings is currently negotiating a contract with a testing laboratory where emissions from their products will be identified and quantified. This will motivate other large manufacturers to undertake similar programs.

A panel from another large manufacturer was recently tested and determined to have very high emissions of a solvent believed to be fairly toxic or irritating to humans. It is a chemical cousin of glycol ether solvents. A 1983 NIOSH current intelligence bulletin warned of its hazards.[24] We have not found definitive toxicity information on this particular product, but its odor alone was sufficient to cause concern.

The molded fiberglass or other acoustic absorbant or barrier material behind the fabric can be a considerable source of VOCs as well as a matrix and food source for microorganisms. Investigation of the contents, emissions, and alternatives should be completed before contracting for office furnishings.

General Recommendations

1. Communicate with manufacturers' technical representatives to obtain the most comprehensive test reports available. If no testing has been done, consider using other products.
2. Evaluate results of testing and adopt mitigation measures based on those results. Consider possible changes in materials, airing-out at the factory prior to packaging, airing-out outside the building prior to installation, or conditioning *in situ*.
3. Consider the need for independent testing. More laboratories are becoming available to do this type of work.
4. Consider random testing of panels arriving on site to monitor emissions.
5. Include specification statements regarding concerns, identification of components, requirements for controlling emissions to minimum levels, and the need to address concerns prior to initiating manufacturing of the panels.

Sponge Effect

When furnishings are installed or finishes are applied in spaces containing high surface area materials, such as carpeting or ceiling tiles or free-standing partitions, much of the initially released VOC will be absorbed onto other material surfaces. The quantities adsorbed will depend on the total surface area exposed as well as the air exchange rate in the space. The rougher surfaces of insulation materials, textiles, and carpets would be expected to adsorb large quantities of VOCs. In fact, the available surface area for adsorption on such "fleecy" materials is many times the plane surface measurement.

When ventilation is reduced or turned off (at night and on weekends, or during warm and cold outdoor temperature periods), indoor air VOC concentrations will tend toward equilibrium; that is, they will rise until there is a balance between emissions from sources and removal from air by ventilation, chemical reaction, or surface adsorption. As VOCs are emitted from materials, they will be adsorbed on surfaces. The higher the temperature, the higher the emissions and air concentrations. The lower the ventilation rate, the more they will be adsorbed on interior surfaces.

An experiment by Berglund et al. demonstrated the "sponge" effect of the distribution of VOCs in enclosed spaces.[3] In the experiment conducted at a Swedish preschool, a stainless steel chamber was lined with floor, wall, and ceiling materials removed from the building. Initial VOC levels in the chamber were similar to those measured inside the preschool, about 100 $\mu g/m^3$. It took 30 days to eliminate the observed sponge effect by supplying 0.5 air changes per hour of outdoor air to the chamber.

Control Measures

The following measures will considerably shorten the time during which the sponge effect will contribute to elevated VOC indoor air levels:

1. Maximum outside air ventilation should be used during and following installation of finishes and furnishings to reduce air levels of VOCs emitted from new products and materials. Use temporary exhaust (through doors, operable windows, stair towers, and emergency exits) for exhausting air rather than the HVAC return system wherever feasible. It is important to operate ventilation systems 24 hours/day, 7/days per week during periods of elevated VOCs.
2. Protect installed materials (with vapor barriers, i.e., sealed plastic coverings) to the extent feasible, during use of VOC-containing finishing products such as adhesives and paints, and during installation of VOC-emitting furnishings and partitions.
3. Protect fiber-lined HVAC ducts and return-air plenums from air flows to avoid contamination of system components. Exposed upper surfaces of ceiling panels and spray-on insulation enclosing concealed spaces used as return air plenums may adsorb large quantities of VOCs if contaminated air is circulated through them.
4. Operate newly occupied building areas at the lowest temperatures acceptable to occupants. Temperature excursions can cause bursts of VOCs with low boiling points and cause episodic elevation of VOC levels.

CONCLUSION

Frequently employed strategies to reduce airborne concentrations of VOCs include several methods: selecting materials with low emissions, treating

materials before use in a building, encapsulating or sealing materials in a building, or by dilution through increased ventilation. A major problem is that little is known about the specific health effects of most VOCs at the low concentrations usually found in indoor environments. Therefore, efforts are not focused on the end point—irritation or toxicity—but rather on the exposure. This is an acceptable approach in light of the absence of sufficient information to target efforts on the active or effective toxic or irritating materials.

APPENDIX

SOURCES OF INDOOR ORGANIC COMPOUNDS

Based on Tucker (ref. 33; see explanatory note at end of this table) and other sources

Compound[a]	Formula	Substantiated Sources[b]	Potential Sources[c]
Formaldehyde	CH_2O	Unvented radiant gas space heaters, upholstery fabric, latex-backed fabric, plywood, particleboard, carpets, paneling, new clothing, fiberglass, paper plates & cups, ceiling panels, airducts, unvented range-top burner, unvented gas oven, urea foam insulation, floor covering, wallpaper, caulking compounds, jointing compound, floor varnish, adhesive, fiberboard, chipboard, linoleum, floor lacquer, calcium silicate sheet, gypsum board, tobacco smoke	
Methylene chloride	CH_2Cl_2	Paint removers, aerosol finishers	
Carbon tetrachloride	CCl_4	Grease cleaners	
Chloroform	$CHCl_3$	Water, clothes washer	
Bromoform	$CHBr_3$		Medicinals
Trichlorofluoro-methane (F-11)	CCl_3F	Refrigerant	
Tetrachloroethylene	C_2Cl_4	Dry cleaning	
1,1,1-Trichloroethane	$C_2H_3Cl_3$	Dry cleaning, cleaning fluid	
Trichloroethylene	C_2HCl_3		Solvent for paints and varnishes, degreasing in dry cleaning
Acetic acid	$C_2H_4O_2$	Tobacco smoke	Food preservative, cooking, solvent for gums, resins, caulks, sealants, glazing compounds, volatile oils
Acetaldehyde	C_2H_4O		Perfumes, flavors, dyes, tobacco smoke
Ethanol	C_2H_6O	Fiberboard	Solvent, antifreeze, tobacco smoke
Isopropanol	C_3H_8O	Particleboard	Antifreeze, solvent for gums, shellac, essential oils, cosmetics

Compound	Formula	Substantiated Sources	Potential Sources
Acetone (propanone)	C_3H_6O	Lacquer solvent	Tobacco smoke
Pyruvic acid	$C_3H_4O_3$		Medicinal ointments
Ethylacetate	$C_4H_8O_2$	Linoleum floor covering	Artificial fruit essences; solvent for varnishes & lacquers; used in manuf. perfumes & artificial leather
Diethylamine	$C_4H_{11}N$		Used in resins, dyes, pharmaceuticals; used in manuf. rubber
Dimethylacetamide	$C_4H_{10}ON$		Solvent for organic reactions
n-Butylacetate	$C_4H_{10}O_2$	Floor lacquer	
i-Butylacetate	$C_4H_{10}O_2$	Floor lacquer	
1,4-Dioxane	$C_4H_8O_2$		Solvent for many oils, waxes, dyes, cellulose acetate
n-Butanol	$C_4H_{10}O$	Edge sealing, moulding tape, jointing compound, cement flagstone, linoleum floor covering, floor lacquer	Flavors, perfumes, industrial cleaners, paint removers
i-Butanol	$C_4H_{10}O$	Edge sealing, moulding tape, jointing compound, cement flagstone, linoleum floor covering, floor lacquer	Tobacco smoke
2-Butanone (MEK)	C_4H_8O	Floor/wall covering, calcium silicate sheet, fiberboard, caulking compounds, particleboard, tobacco smoke	Synthetic resins, tobacco smoke
2-Ethoxy-ethanol (Cellosolve)	$C_4H_{10}O_2$	Epoxy paint, latex paint, polyurethane varnish	
Pentane	C_5H_{12}	Tobacco smoke	
1-Amyl alcohol	$C_5H_{12}O$		Solvent in organic synthesis
Propyl acetate	$C_5H_{10}O_2$		Flavors, perfumes, plastics
n-Hexane	C_6H_{14}	Chipboard, gypsum board, insulation board, floor covering, wallpaper, tobacco smoke	
Cyclohexane	C_6H_{12}	Tobacco smoke	Solvent for lacquers and resins, paint & varnish remover
Cyclohexanone	$C_6H_{10}O$		Solvent for many resins and for DDT, also for fats & waxes
Hexanal	$C_6H_{12}O$	Polyurethane wood finish	
4-Methyl-2-Pentanone	$C_6H_{12}O$	Floor/wall covering, tobacco smoke	
Methylcyclopentane	C_6H_{12}	Tobacco smoke	

Compound	Formula	Substantiated Sources	Potential Sources
2-Methylpentane (isohexane)	C_6H_{14}	Chipboard, gypsum board, insulation foam, floor covering, wall paper, tobacco smoke	
3-Methylpentane	C_6H_{14}	Tobacco smoke	
Benzene	C_6H_6	Smoking, adhesives, spot cleaners, paint remover, particleboard, tobacco smoke	
Chlorobenzene	C_6H_5Cl		Solvent for paint; used in manuf. DDT & phenol
o-Dichlorobenzene	$C_6H_4C_{12}$	Deodorizers, moth crystals	
m-Dichlorobenzene	$C_6H_4C_{12}$	Deodorizers, moth crystals	
p-Dichlorobenzene	$C_6H_4C_{12}$	Deodorizers, moth crystals	
Hexachlorobenzene	C_6Cl_6	Fungicide	
Tetrachlorophenol	$C_6H_2Cl_4O$	Wood preservative	
2-Ethoxyethylacetate	$C_6H_{12}O_3$	Floor lacquer, epoxy paints	
Pentachlorophenol	C_6HCl_5O	Wood preservative, disinfectant, fungicide	Paints, wallpaper adhesive, textiles, wood finishes, leather tanning, canvas, rope, paper, carpet shampoo
Iso-butylacetate	$C_6H_{12}O_2$		Flavorings, solvent
Butylacetate	$C_6H_{12}O_2$	Floor lacquer	
Toluene	C_7H_8	Solvent-based adhesive, water-based adhesive, edge sealing, moulding tape, wallpaper, jointing compound, calcium silicate sheet, floor covering, vinyl coated wallpaper, caulking compounds, paint, chipboard, linoleum floor covering, kerosene heaters, tobacco smoke	
Butyl acrylate	$C_7H_{11}O_2$		Used as monomer in manuf. of polymers & resins for textile & leather finishes
Heptane	C_7H_{16}	Floor covering, floor varnish, kerosene heaters	
Benzaldehyde	C_7H_6O	Fiberboard, particleboard	
Ethylbenzene	C_8H_{10}	Floor/wall covering, insulation foam, chipboard, caulking compounds, jointing compound, fiberboard, calcium silicate sheet, adhesives, floor lacquer, grease cleaners	
Styrene	C_8H_8	Insulation foam, jointing compound, fiberboard, tobacco smoke	

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Compound	Formula	Substantiated Sources	Potential Sources
Xylenes	C_8H_{10}	Adhesives, jointing compound, wallpaper, caulking compounds, floor covering, floor lacquer, grease cleaners, shoe dye, tobacco smoke, kerosene heaters, varnish, kerosene heaters	
Nonane	C_9H_2O	Wallpaper, caulking compounds, floor covering, chipboard, adhesives, cement flagstone, jointing compound, floor varnish, kerosene heaters, floor wax	
Ethyl toluene	C_9H_{12}	Floor wax	
o-Ethyltoluene	C_9H_{12}	Floor wax	
m,p-Ethyltoluene	C_9H_{12}	Floor wax	
m-Ethyltoluene	C_9H_{12}	Floor wax	
Quinolone	C_9H_7N		Used in manuf. of dyes; solvent for resins
Isoquinolone	C_9H_7N		Used in synthesis of dyes and insecticides, rubber accelerator
Indane (hydrindene)	C_9H_{10}		Constituent of coal tar
1,2,3 Trimethylbenzene	C_9H_{12}	Floor/wall covering, floor wax	
1,2,4 Trimethylbenzene	C_9H_{12}	Floor/wall covering, linoleum floor covering, caulking compounds, vinyl coated wallpaper, jointing compound, cement flagstone, floor varnish, chipboard, floor wax	
1,3,5 Trimethylbenzene	C_9H_{12}	Caulking compounds, floor/wall covering, floor wax	
n-Propylbenzene	C_9H_{12}	Adhesives, floor/wall covering, chipboard, paint, caulking compounds, insulation foam, kerosene heaters	
n-Butylbenzene	$C_{10}H_{14}$		Solvent
Limonene	$C_{10}H_{16}$	Paint, adhesives, chipboard, detergents	
Pinene	$C_{10}H_{16}$		Used in manuf. of camphor, insecticides, solvents, plasticizers, & perfumes
a-Pinene	$C_{10}H_{16}$	Cement flagstone, fiberboard, gypsum board, adhesive, insulation sheets, chipboard, calcium silicate sheet	
a-Terpinene	$C_{10}H_{16}$		Oil of lemon
Camphene	$C_{10}H_{16}$		Occurs in many essential oils
Camphor	$C_{10}H_{16}O$	Moth crystals	
Naphthalene (Tetralin)	$C_{10}H_{12}$	Moth crystals	

Compound[a]	Formula	Substantiated Sources[b]	Potential Sources[c]
Undecane	$C_{11}H_{24}$	Wallpaper, gypsum board, floor/wall covering, joint compound, chipboard, floor varnish, paints, paint removers	
Dodecane	$C_{12}H_{26}$	Floor varnish, floor/wall covering, kerosene heaters	
4-Phenylcyclohexene	$C_{12}H_{14}$	Latex-backed carpet	
Nonylphenol isomers	$C_{15}H_{24}O$		Used in manuf. of lubricating oil additives, resins, plasticizers, & surface active agents
Dibutylphthalate	$C_{16}H_{22}O_4$	Plastics	

NOTES:
[a] Selected compounds that have been measured in indoor air and that may have come from material sources.
[b] Source types for which quantitative data on emissions have been obtained by chamber tests, or for which qualitative data are available (e.g., from headspace testing).
[c] Source types known to contain the compound. Not all products of the source type will necessarily have the compound, however.

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Building Commissioning

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Building Commissioning

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Abstract

In order to detect and correct defects in a timely, cost effective manner, HVAC system commissioning has recently been used as part of building construction. Preliminary evidence suggests that proper and complete commissioning is cost effective. It is probable that more comprehensive building commissioning will become standard construction practice during the next few years.

Introduction

Problems in newly-completed or remodeled structures have created awareness that traditional building close-out procedures are not sufficiently sensitive to detect defects in building components and operation. Investigators often discover major defects in HVAC system design, construction or system balancing during investigations of occupant complaints (Levin and Phillips 1989).

HVAC System Commissioning Costs and Benefits

The costs of discovering and remedying HVAC system defects are substantial. In addition to the costs of investigation and remediation of problems, they have included costs attributable to occupant illness and absence from work; meeting time for owners, designers, contractors, employers, and occupants; and, lost revenues for landlords and tenants. The British Columbia Buildings Corporation has studied the costs and estimated avoided costs of HVAC commissioning, and it has concluded that HVAC system commissioning is cost effective (present value = \$0.20 to \$0.50 per square foot cost savings over the first five years). Table 1 is a summary of the analysis (Levin, 1989).

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Table 1. Economics of HVAC Commissioning

Item	$/Sq Ft Based on HVAC	Comment
A. COSTS FOR COMMISSIONING:		
Designer	$0.02 - 0.10	Additional time spent on site to witness and verify functional performance testing.
Contractor	$0.10 - 0.20	Direct commissioning activities. If documented at beginning, results in no additional costs for construction and avoids call-backs.
Owner	$0.025 - 0.10	Additional involvement of operational staff during construction: familiarization with systems.
SUBTOTAL	$0.145 - 0.40	
B. AVOIDED COSTS:		
Energy	$0.13 - 0.26[a]	Shortens usual 3 year period for building operators to learn optimal energy operation
Maintenance	$0.15[b]	Estimated reduction due to proper initial functioning of equipment and training of operational staff; present value for 5 years' savings.
Construction	$0.07	Correction of construction problems defects by contractor prior to occupancy and at no cost to owner.
Satisfied tenants	$0.25[d]	Avoiding meetings to resolve tenants' problems, avoiding occupants' absence due to illness.
SUBTOTAL	$0.60 - 0.76	

C. NET RESULT: $0.20 - 0.515 / Sq Ft savings over first five years.

NOTES:
a Present value calculation
b Estimated from actual cost data
c Based on studies of 5 buildings, 2 had serious problems, costs averaged over 5 buildings.
d Based on the following assumptions:
Every fifth person in meetings 30 min./mo. = $0.10/yr/sq ft
Owner meetings and direct costs = $0.05/yr/sq ft
Every fifth person out of work (illness) 2 days/year = $0.25/yr/sq ft.

Why HVAC Systems Commissioning?

Many indoor air quality problems have occurred during initial occupancy of newly-constructed or remodeled buildings. Some of these problems have been directly attributed to incomplete or improper construction, especially of HVAC systems (Levin 1989b; Trueman 1989). Some of these problems would be discovered by a thorough and effective commissioning process.

Large construction and development firms currently use HVAC systems commissioning as a normal construction activity. According to an executive of a major U. S. development firm, if clearly documented in the construction contract and followed during implementation, HVAC system commissioning does not cost any more at all (Brickman, 1989). And the benefits of commissioning warrant the extra effort by all parties (Trueman 1989).

In fact, commissioning is not new. The concept of HVAC commissioning has been used effectively in Europe and Canada for a long time. It is a wonder that it has not be utilized more extensively and routinely in the United States.

Guidance for HVAC System Commissioning

The American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc. (ASHRAE) is developing guidelines for HVAC system commissioning in response to the growing professional interest. A Public Review Draft Guideline for Commissioning HVAC Systems was released in June 1988 (ASHRAE 1988), and adoption of the final document is expected in June of 1989 (Levin 1989). The draft Guideline identifies the roles of the several designers, contractors and other parties. A series of papers by members of the ASHRAE committee preparing the document presents an elaboration of the outline in the draft and indicates what the final document might contain (Brickman 1989; Gill 1989; Lawson 1989; Stone 1989; Trueman 1989).

What Is HVAC System Commissioning?

The process involves comprehensive planning, thorough documentation and systematic implementation. Another way of thinking about it is that proper commissioning is merely building it right in the first place and making sure that it works right before occupying the building.

The HVAC system commissioning process requires clear descriptions of the following:

* Sequence of actions involved in commissioning.
* Performance required by the system(s) to be commissioned.
* The intended use and operation of the system(s).
* The responsibility of all entities for commissioning activities.

Suggested components of an HVAC system commissioning program are described in Table 2 below.

Table 2. HVAC System Commissioning Process

Design phase (by architect/engineer)
* Establishment of clear design criteria
* Documentation of HVAC design criteria and systems description (see Table 3)
* Preparation of a commissioning plan
* Describe verification procedures
* Define documentation requirements for commissioning process including all reports, submittals, drawings, schematics, checklists, operating data, maintenance data, and as-built documentation.

Construction phase
* Pre-commissioning preparation for start-up
  - Personnel selection
  - Pre-commissioning meeting of designer, owner, and contractor representatives
* Actual system start-up: initial operation of all equipment
* Final start-up -- complete performance inspection
  - Temperature control system
  - Facility automation system
  - Testing and balancing
  - Equipment documentation

Final commissioning
* Meeting of all relevant parties to discuss system and answer any questions about system sequences, set points, operation; review all final documentation for submittal to owner.
* Assemble all documents for submittal to owner
* Train operational personnel in the following:
  - System philosophy
  - System familiarization
  - System sequence
  - System maintenance
  - System diagnosis
  - Facility automation system

Design Documentation

A major requirement of HVAC system commissioning is clear, specific and complete design documentation. It forms the basis for identifying the components to be commissioned, the design or performance standards, the operational modes, the loads to simulate, and other essential design criteria and system descriptions. ASHRAE's draft revised ventilation standard 62-1981R also requires design documentation for its implementation (Levin 1989). A suggested set of elements to be included in the design documentation is listed in Table 3.

Table 3. Suggested Elements of Design Documentation

A. DESIGN CRITERIA:

1. Indoor design conditions (all seasons): Temperature, relative humidity, air movement.
2. Outdoor design conditions (all seasons): Temperature, humidity, wind direction and velocity, location and timing of outdoor sources of air pollutants of concern.
3. Assumed or anticipated occupant densities, activities and use patterns for each space or type of space in the building.
4. Assumed electrical load for light and power.
5. Any special loads which might exist.
6. Outside air supply rates under various operating conditions and loads.
7. Assumed ventilation effectiveness for each type of space under HVAC system mode of operation which result in differing supply design conditions. Assumed distribution characteristics for each ventilation or air circulation condition including conditions of minimal air circulation and of upper and lower supply air temperature limits.
8. Definition of building envelope, including type and characteristics of materials and assumed infiltration.
9. Air quality design criteria.
10. Code requirements
11. Noise criteria.
12. Fire and safety requirements
13. Energy efficiency and projected operating cost.

B. HVAC SYSTEM DESCRIPTION:

1. Basic system types
2. Major components
3. Capacity and sizing requirements
4. Redundancy provisions
5. Intended operation in each seasonal mode, including designed changeover conditions.
6. Changeover procedures.
7. Part-load operational strategies for each season.
8. Occupied/unoccupied operation modes for each season.
9. Design setpoints for control system, including permissible limits of adjustments.
10. Operation of system components in life safety modes.
11. Energy conservation procedures.
12. Any other engineered operational mode of the system.

Extending Commissioning to the Whole Building

Building commissioning most commonly focuses on HVAC equipment, but some building designer, developers, and owners have extended the commissioning process to non-HVAC system materials, equipment and other building elements. Surely the plethora of technology involved in "smart" buildings warrants a thorough shakedown before the final payment is issued and the building is occupied.

Life safety systems, communications systems, transportation systems, security systems, and other "high tech" devices must be tested in place under simulated loads and real situation use conditions to initiate the warranty period and be approved for use. While commissioning appears most applicable to building equipment, it may also be applied to other building components such as thermal or moisture protection systems, shading devices, expansion joints, radon mitigation measures, and others.

Conclusion

The commissioning of HVAC systems should be standard practice in major building contracts. It is in everyone's interest. The questions that occur are 1) Will HVAC system commissioning become standard practice? and, 2) Will HVAC system commissioning be expanded to cover all components and systems in new buildings in the future? The "smart" builder will answer "yes!"to both questions.

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SICK BUILDING SYNDROME:
REVIEW AND EXPLORATION OF CAUSATION HYPOTHESES AND CONTROL METHODS

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ABSTRACT

Control and abatement of indoor air quality (IAQ) problems are dependent upon reliable investigation and diagnosis. Sick building syndrome (SBS), building related illness (BRI), and other health and comfort problems are selectively reviewed and discussed. Psychological and social as well as physical, chemical, and biological factors that affect occupant physiological and health responses are identified.

Confusion exists regarding definitions and attributes of problem buildings. Timely, comprehensive, systematic investigations are rare, expensive, and difficult. Systematic and other biases result in inadequate investigations and incomplete or incorrect diagnoses. Building ecology and building diagnostics are described as a comprehensive framework for understanding and investigating indoor air quality problems.

Hypothesized causes of SBS are identified based on published SBS and BRI investigation reports and review articles. Methods to control SBS, BRI, and other building-associated illnesses are presented and discussed. Preventive measures to control IAQ-related health and comfort problems and recommendations for further research are given.

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SICK BUILDING SYNDROME: REVIEW AND EXPLORATION OF CAUSATION HYPOTHESES AND CONTROL METHODS

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INTRODUCTION

Sick building syndrome (SBS) may affect as many as 20% of the office workers in the United States. In a survey of U.S. office workers, symptoms associated by respondents with poor air quality included a tired, sleepy feeling (56%); a congested nose (45%); eye irritations (41%); difficulty in breathing (40%); and headaches (39%) (Woods 1987). Efforts to control and abate the causes of SBS in buildings are potentially important to the economy and to public health. The present paper is a review of selected published reports related to SBS and a discussion of some of the problems limiting our understanding of it.

The complexity of modern buildings presents significant unmet challenges to designers, operators and investigators. Problems other than air quality can cause or exacerbate the symptoms of sick building syndrome. Psychological and social as well as physical and biological factors interact to create occupant physiological and health responses to building environments. Yet detailed, comprehensive investigations of building-associated outbreaks are infrequent due to the resources and personnel required to conduct them.

Control and abatement of SBS is dependent upon knowledge developed through reliable investigations and diagnoses. Understanding the potential causes of SBS is essential to such investigations and diagnoses. No clear understanding of SBS and no consistent definition of SBS is used. Authorities in the field use differing definitions or confusing terms which impede progress in understanding the phenomenon. In fact, the most widely accepted definition of SBS requires the absence of identified causes, but even those who present this definition fail to use it consistently.

Knowledge and understanding of SBS is obtained through four primary means: (1) investigations of problem or complaint buildings, with or without non-complaint control buildings; (2) multiple building studies, which may or may not include complaint buildings; (3) controlled experiments in buildings or laboratories where environmental factors are manipulated and the responses of occupants are surveyed; and (4) literature reviews where data or findings from various investigations or studies or both are collected and analyzed. The investigations are usually commissioned by building owners, operators, or occupants while the studies and research are usually funded by public or private research or by governmental agencies. Reviews (including the present paper) are usually initiated by interested authors.

DEFINITIONS OF SBS AND OTHER BUILDING-ASSOCIATED ILLNESSES

SBS is variously defined by its symptoms, by its hypothesized causes, or by the demonstration of a statistically valid association of SBS symptoms with a particular building.

Symptoms

SBS is frequently defined by the occurrence of reported symptoms from a group of symptoms listed in several authoritative publications. Most of these definitions declare that the symptoms abate upon leaving the building and worsen upon re-entry (NAS 1981; WHO 1983; Stolwijk 1984; Fanger 1987; Finnegan et al. 1984; Molhave 1987; Woods 1987). A widely cited World Health Organization report (WHO 1983) lists a broad spectrum of symptoms reported primarily in Scandinavia and the United States. Those symptoms have many features in common and are listed in Table 1.

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Molhave (1987) has proposed a classification scheme for the major *symptoms of SBS (Table* 2). A World Health Organization Working Group on Indoor Air Quality Research (WHO 1986) has proposed a classification scheme for "sick" buildings (Table 3). These two authorities have presented conflicting schemes. Their lists are inconsistent, and Molhave has classified SBS symptoms while WHO has classified "symptoms found in sick buildings." The WHO list includes symptoms which it states are not found in sick building syndrome, so a sick building is not equated exactly but overlaps with one in which sick building syndrome occurs.

Causal Factors

Commonly, several broad classes of factors are considered potentially related to an elevated incidence of reported symptoms when a problem is termed SBS (Skov and Valbjorn 1987; Molhave 1987). These factors include chemical (Molhave et al. 1984), physical (Alsbirk 1983), biological (Morey 1984), and psychosocial factors (Colligan 1981; Alexander and Fedoruk 1986).

Many authors state that most investigations of SBS have not resulted in definitive identification of causal factors. In fact, most definitions of SBS require that the reported symptoms not be associated with specific environmental or other causal agents. Other definitions of SBS expressly require that the causal agent(s) not be clearly identified or demonstrated by the investigation. Generally, where a causal agent is identified, the symptoms are no longer considered "sick building syndrome"; rather, the building problem is specified as contamination by the causative agent and the illness is termed building-related illness (see discussion below). But confusion exists because many investigators still apply the term SBS to cases where symptom etiology is clearly identified.

Elevated Rate of Symptom Reports

Some definitions of SBS require the demonstrated presence of excess reported symptoms in the complaint building compared with a control building or some other comparable baseline. This involves surveys of building occupants, usually using epidemiologic techniques. However, the measurements made in most reported studies are insufficient to enable identification of the pollutant concentrations and their associations with symptoms. There are usually too few measurements of environmental factors. Large occupant populations are studied through questionnaire surveys, either self-administered or administered by the investigators. The large expense of comprehensive pollutant measurements in each distinct environmental niche within a study building results in few samples of few pollutants in nearly all investigations. Large spatial and temporal variability of indoor air pollutant concentrations within a single building or even spaces within the building (for CO_2, RSP and VOC, for example) reduce the likelihood that one or a small number of measurements will provide adequate information to identify associations between exposures and symptoms. The "limitations of investigations" are explored later in this paper.

Under the elevated symptom prevalence definition, one or even a small percentage of a building's occupants cannot suffer from sick building syndrome regardless of the symptoms or the building-relatedness of their onset and recurrence.

Sources of Confusion About Terminology

There are two sources for much of the confusion. One involves definitional differences; the other involves inconsistent use of terms. Where the etiology of symptoms and complaints is identified, most investigators do not label the symptoms or complaints SBS or the building "sick" (WHO 1983; Molhave 1987; Woods 1987). Rather, they define the symptoms as manifestations of illness and classify it as building related illness (BRI) to distinguish it from SBS. BRI includes such medical conditions as hypersensitivity pneumonitis, Pontiac fever, and allergic dermatitis (Hodgson 1986; Woods 1987). Building-related illnesses are attributed to a broad range of pollutants including infectious microorganisms, allergens, chemicals, moisture, temperature, noise, vibration, and poor illumination.

Building-Associated Epidemics
Kreiss and Hodgson have used the term "building-associated epidemics" to include both SBS and BRI (Kreiss and Hodgson 1984). Other authors have used BRI as the all-inclusive term covering two categories of episodes: "those characterized by a generally uniform clinical picture for which a specific etiology can often be identified, and those in which affected workers report nonspecific symptoms temporally related to work" (Samet *et al.* 1988).

While asserting that SBS does not involve identified etiology, some authors still label cases as SBS which include bacterial diseases (such as Legionnaire's disease and Pontiac fever), thermal discomfort, and irritation caused by chemicals, and they call such buildings examples of "sick" buildings (WHO 1986; Berglund and Lindvall 1987). Thus, confusion is caused by the overlapping but different use of the terms "sick building" and "SBS."

Some authorities argue that buildings cannot be "sick," they may only be contaminated. The term "sick building syndrome" may just inappropriately medicalize an engineering, architectural, or maintenance problem (Hodgson 1989).

Sick Buildings
In listing types of "sick buildings," Berglund and Lindvall (1986) include buildings contaminated with radon, molds, contagious agents, and formaldehyde, and they add to the list "buildings in which the occupants show reactions and symptoms similar to those known to be caused by formaldehyde (Andersen et al. 1975) but in which the concentrations of formaldehyde are well below known reaction thresholds." Berglund and Lindvall appear to focus on defining "sick" buildings rather than the symptoms of the occupants, as in SBS. Recently, Berglund and Lindvall have promoted use of the term "healthy buildings" through the convening of an international symposium on the subject (Berglund and Lindvall 1988).

Alexander and Fedoruk (1986) have categorized the particular type of problem building added by Berglund and Lindvall as "epidemic psychogenic illness" or "mass hysteria." According to Alexander and Fedoruk, the terms are interchangeable but the first is preferred due to the tendency to misunderstand the second. Like many investigations of sick building syndrome, the diagnosis of mass psychogenic illness is difficult to document and strongly resisted by the affected building occupants (Alexander and Fedoruk 1986).

An example of mislabeling is a recently reported investigation of "tight building syndrome," or "closed building problem," and "new building problem." The authors described the case as fitting the WHO (1983) SBS definition including the usual symptoms and the failure to identify the causative agent(s) (Whorton et al. 1987). When an extended summary of the Whorton article was prepared by others later, the case was termed an investigation of an outbreak of "building related illness" (HESIS 1987).

Building-Related Illness
Many authors explicitly distinguish SBS from building-related illness (BRI),which includes allergic respiratory disease (sinusitis, tracheobronchitis, asthma, hypersensitivity pneumonitis, and humidifier fever), skin diseases (irritant, allergic, and photodermatitis) irritant syndromes (carpet shampoo, formaldehyde), and infections (Legionnaire's disease, Pontiac fever, Q fever) (Kreiss and Hodgson 1984; Hodgson and Kreiss 1986; Stolwijk 1984; Woods 1987; Molhave 1987).

Building Sickness
The term "building sickness" was proposed in 1984. Lars Molhave of Denmark suggested the term "building sickness" to characterize reported symptoms in a building in which "the occupants report comfort or health problems which they assign to the indoor atmospheric environment." Molhave would limit the use of the term "sick building" to cases in which the problem is identified as multifactorial and in which no measured factor exceeds generally accepted thresholds or recommendations (Molhave 1987). This is similar to most definitions of SBS without the criterion that there be a statistically significant excess of reported SBS complaints and symptoms.

It is possible that the term "building sickness" has not received more use due to the alternate meanings of the initials BS -- British Smoke, which is used in epidemiologic studies involving particulate matter, and a vulgar American slang expression.

Etiology of Confusion

Molhave suggests that the different or inconsistent use of terminology results from the involvement of different groups of "indoor climate experts." For example, the term "irritation" is used by medical experts as a synonym for toxic skin damages known from occupational exposures; by technical and engineering experts to describe acceptability or unacceptability of the indoor environment; and by the occupants to describe subjective feelings of reduced comfort due to dry nose, dry eyes, and dryness or stuffiness of the air (Molhave 1987). Thus, the backgrounds of the investigators may significantly impact the diagnoses.

Working Assumptions

In the remainder of this paper, the definitions and concepts articulated by the WHO Working Group (WHO 1983), Stolwijk (1984), Molhave (1987), and Woods (1987) are relied upon as the basis for the discussion. These exclude building-related illness and emphasize multifactorial sick building syndrome.

Classification of the occupants' conditions in problem buildings will be classified according to a scheme adapted from that proposed by Woods as follows: 1) unresolved building-related complaints or symptoms; and, 2) building associated illness including a) building sickness, b) building-related illness, and, c) unclassified building-associated illness. Woods has also added the category "undetected problem buildings" which will not be discussed further here (Woods 1988). See Table 4 for definitions of each of these categories.

FINDINGS FROM MAJOR STUDIES

Potential Etiologic Agents of SBS from Recent Studies

Wallace has reviewed the recent literature on SBS (1988). The review covers published reports from 1984 through 1987. The results of his review are presented in Table 5. Five causal factor categories were developed: physical, chemical, biological, psychological, and multifactorial. Wallace did not review research on psychological factors.

The causal factors hypothesized cover a wide range and the study methods also were diverse. Wallace drew no conclusions from the literature, but his presentation is interesting for the range of factors considered and the amount of interest in SBS represented by the reviewed work.

Several major multi-building, multi-disciplinary studies have been reported, four in Great Britain (Finnegan et al. 1984; Pickering et al. 1985; Harrison et al 1987; Hedge et al 1987)) and one in Denmark (Skov and Valbjorn 1987). They have developed conflicting conclusions about SBS causal factors.

Two British studies found higher symptom prevalence rates in mechanically ventilated buildings than in naturally ventilated ones (Finnegan 1984; Harrison 1987). Among the mechanically ventilated buildings in the later study, those with humidification had the highest symptom rates. However, among 11 naturally ventilated buildings were two in which symptom prevalence rates were typical of the 16 sealed buildings in the study (Harrison et al. 1987).

Finnegan reported a threefold excess of SBS symptom prevalence rates in five mechanically-ventilated buildings (15%-45%) compared with three naturally ventilated buildings (5%-15%) (Finnegan and Pickering 1987). There was a noticeably wide range of building-specific symptom prevalence rates (5%-45%) in buildings characterized by the authors as non-complaint buildings. Incomplete evaluation of building system performance or operation through either inspection, analysis, or environmental measurements has been reported by these authors. No reported measurements have been published.

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Another large scale British study of 30 buildings found "few differences in symptom prevalence" between naturally and mechanically ventilated buildings. (The definition of mechanical ventilation here was "a ducted system but with no plant for heating and cooling.") Higher symptom prevalence rates were found for all symptoms in air-conditioned than in non air-conditioned buildings and for females than for males. A higher prevalence was found for all symptoms and all ventilation system types in the public sector than in private-sector buildings (Hedge 1987). The published report does not detail the inter-building variations in prevalence rates.

The Danish investigators found no correlation between complaints and building ventilation type. They found a number of factors associated with elevated symptom prevalence including age of building, sex of occupant, job category, type of work, temperature, number of occupants, amount of open shelving, and what they called the amount of "fleecy material" (Skov and Valbjorn 1987). These findings are discussed in greater detail below.

"Multifactorial Sick Building Syndrome"

Several investigators have suggested that the etiologic agents in SBS were multiple factors, none of which alone causes increased symptoms and complaints (Turiel et al. 1983; Molhave 1987; Woods 1988; Valbjorn and Skov 1987). In fact, measurement of a broad spectrum of environmental parameters usually fails to isolate particular agents as etiologic. Multifactorial analysis identifies clusters of factors associated with higher rates of reported symptoms (Valbjorn and Skov 1987).

The Danish Town Hall Study

The most comprehensive SBS study reported to date is a multifactorial investigation known as the Danish Town Hall Study (DTHS). Covering a total of 27 buildings that were not known problem buildings, the DTHS involved measurements of indoor climate and other environmental parameters in 14 town halls. A questionnaire and clinical study of 4369 employees in the town halls and 13 affiliated buildings was also conducted. While reported symptom levels were high for mucosal irritation (28%) and for general symptoms in the form of headache, abnormal fatigue, or malaise (36%), the measurements of environmental parameters did not result in elucidation of the epidemiology.

However, the differences in the prevalence of symptoms among buildings was significant and was correlated with building factors as well as occupant factors. Building factors that could explain the difference in the prevalence of symptoms included the total weight and potentially allergenic fraction of floor dust, the area of "fleecy" material per cubic meter of air, the length of open shelves per cubic meter of air, the number of work stations, and the air temperature. Occupant factors included sex, type of work, and job category. See Table 6 for the results of the environmental measurements (Valbjorn and Skov 1987).

Among the findings were the following:

1. Elevated rates of reported mucosal irritation was associated with the size of the allergenic fraction of floor dust, the length of open shelves per cubic meter of air, the area of fleecy material per cubic meter of air, the number of work stations, and air temperature.

2. Symptoms correlated strongly with job category. The symptom prevalence varied highly with job category, and the highest prevalence was found in the subordinate job categories. Jobs involving photoprinting, working at video display terminals, and handling carbonless paper correlated with the reported frequency of mucosal irritation and of general symptoms; the number of weekly working hours of women also correlated with reports of these two symptom categories, although less markedly.

3. As in several other studies, women had a higher symptom prevalence rate than men and complained more frequently about indoor climate.

4. Symptom prevalence rates varied significantly among buildings, supporting the notion that the symptoms are building-related. Individual town halls correlated significantly with reported mucosal irritation and general symptoms. The lowest prevalence of symptoms was found in the oldest town halls (buildings were mostly newer than 30 years of age, with one almost 50 and another 80 years old).

5. The difference between mechanically and naturally ventilated buildings was not significant for this study. This is in sharp contrast to the findings on the large-scale British study (Pickering et al. 1984; Robertson et al. 1985).

The DTHS strongly supports the findings of many investigations that extensive measurement of environmental variables, including air quality, may not reveal "the cause" of the complaints. It appears more likely that a constellation of factors including some combination of those identified above as well as chemical or biological contamination and improper ventilation system design, construction, maintenance, or operation will be found in problem buildings.

POTENTIAL ETIOLOGIC AGENTS IN SBS FROM THE DTHS

We have hypothesized etiologic relations of some potential causal agents/factors identified in the Danish Town Hall Study (see Table 7). In Table 8 we have attempted to identify potentially additive or synergistic co-variables. The following discussion is based on those two tables. The discussion focuses on some environmental and institutional factors frequently associated with increased symptom or complaint rates in problem buildings (Turiel 1983; Molhave 1987; Valbjorn and Skov 1987; Woods 1987).

VOC as Potential Sources of Complaints

Reporting on an earlier study, Molhave (1982) identified 42 commonly used building materials and measured their volatile organic compound (VOC) emissions. A total of 52 compounds were identified. An average of 22 compounds was identified from each material, and the range of emission rates was extremely large. The arithmetic average emission rate was 9.5 mg/m^2h. Three model rooms constructed from the materials were found to contain between 23 and 32 of the compounds at concentrations from 1.6 to 23.6 mg/m^3. When the cancer risks and health effects (Molhave classifies irritation as a health effect) of each of the 52 compounds was reviewed, 82% were known or suspected mucous membrane irritants and 25% were suspected or known animal carcinogens.

A very high percentage of common VOC emitted from building materials is known or suspected mucous membrane irritants; therefore, it is reasonable to expect significant numbers of building occupants to experience mucous membrane symptoms in newly constructed, remodeled, or furnished buildings. Turiel et al. (1981) suggested that a number of contaminants acting synergistically may have been responsible for the higher symptom incidence in a comprehensively investigated problem building. Hollowell (1981) suggested that the reason building occupants complained about what they ought not to be able to perceive (VOC) at the very low measured airborne concentrations was that the composite effect might give rise to the reported health effects. Others have supported that theory (Molhave 1982; Stolwijk 1984).

Noma et al. (1988) have found correlations between VOC profiles and their distribution patterns in one "sick" and one "healthy" Swedish preschool of identical design. Using sophisticated statistical analyses, they examined the patterns of VOC distribution in various locations in the two buildings. They concluded that the distribution of VOC was more uniform in the healthy than in the sick preschool.

Thermal Factors

Elevated temperature in a building can have many effects on the building environment and directly or indirectly on the occupants. Not the least among them is discomfort from the temperature itself. This discomfort can reduce tolerance to other factors, many of which may be exaggerated by the elevated temperature. Additionally, microorganism growth may be enhanced, VOC emissions from materials will increase, and ventilation airflow will normally decrease.

Microorganism contamination. Biological aerosol concentrations might increase due to increased growth and proliferation of microorganisms associated with higher temperatures, reduced outside air flow, and increased demand on air-conditioning equipment. Some organisms may proliferate outdoors or on building equipment surfaces in warmer weather. An important example are the legionella bacteria responsible for Pontiac fever, which has been reported as occurring almost exclusively in spring and summer (Friedman et al. 1987). Many of the reported outbreaks of Legionnaire's disease have also occurred in spring or summer.

VOC Emissions. VOC emissions will increase as a result of the temperature-based increase in vapor pressure. Girman (1987) has calculated that a 13^0C rise in temperature will result in a 200% increase in typical VOC vapor pressure. The increase in emissions will be greatest from materials with large surface areas in the air stream and from materials where the emission process is dominated by evaporation from the surface rather than diffusion from within the material. Some of the materials with large exposed surface areas are freestanding partitions, bottoms of ceiling tiles facing the interior, tops of ceiling panels facing concealed spaces serving as return air plenums, fibrous linings of air ducts, and textiles or fabrics covering walls, furnishings, or floors.

Ventilation Airflow. Many mechanical ventilation systems will reduce airflow and outside air supply to the interior when temperatures rise toward the upper end of the comfort range. This is particularly true of variable-air-volume (VAV) systems. When temperatures are elevated, increased ventilation is most necessary to remove contaminants resulting from higher emission rates and increased airflow is required to provide evaporative cooling of occupants' exposed skin surfaces. Yet, under most ventilation system control designs, airflow or ventilation or both may be reduced.

High Surface Area Interiors

"Fleecy material" and "open shelves" were identified as risk factors in the Danish Town Hall Study. "Fleecy" refers to materials such as fabrics and carpets which have rough, textured surfaces. The association with elevated symptoms might result from the extremely large effective surface areas facing the interior space on fleecy materials or open shelving systems. "Effective" surface area refers to the actual surface area available for adsorption and re-emission sites for VOC or deposition of small particles. Recent advances in mathematics (fractal math) have shown that the actual surface area available for particle deposition or molecular adsorption is many times larger than the two-dimensional (plane geometry) measured surface area. Small particles, which penetrate deepest in the respiratory system, deposit equally efficiently on horizontal and on vertical surfaces (Weschler 1988).

Carpets, textiles used for wall coverings and furnishings, and insulation materials facing the interior or the air stream in mechanical ventilation systems are high-surface area materials that provide more adsorption sites for VOC and more deposition sites for small particles. Fibrous materials also provide readily available source material for airborne particles through surface erosion, abrasion, or deterioration of the binding forces. Housekeeping tasks including cleaning, vacuuming, and dusting are made more difficult by rough surfaces and the larger surface areas, thereby resulting in the presence of a greater reservoir of unattached particles which may become airborne when disturbed by human activity or ventilation airflow.

VOC Emissions. VOC emitted from building materials have been shown to distribute themselves on exposed materials throughout enclosed spaces and then are re-emitted for several weeks or more (Berglund et al. 1987). Many "fleecy" materials (such as carpets, upholstered furnishings, fabric wall coverings, fiberglass insulations and air ducts) are known sources of

VOC. Many carpets and wall coverings are fastened to the floors and walls, respectively, with adhesives which are known sources of VOC. It is apparent that buildings with large surface areas (from both fleecy materials and open shelves) will likely be associated with elevated VOC air concentrations. It is also likely that VOC concentrations in such buildings will decrease more slowly than in buildings with hard or smooth surfaces and less surface exposed to the interior.

Age of Building

A clear association was found in the Danish Town Hall Study between age of building and complaint or symptom rate, with the oldest buildings having the lowest rates. Newer offices are often constructed from softer, less durable materials on the major surfaces -- floors, walls, and ceilings. This could result in higher airborne particle concentrations from deterioration of the surfaces or finishes and polishes applied to them. Newer offices are usually planned with some or all "open office planning" rather than predominantly enclosed or private office spaces typical of older offices.

Densities of workers (per unit of area) in open offices are usually higher than in private offices. This results in many environmental problems, including noise; chemical, physical, and biological contamination of air; lack of visual privacy; lack of audial privacy; and lack of control over personal workspace.

Architects usually control open office acoustic problems by using high-surface-area materials and components (open shelves, freestanding partitions) and by utilizing fleecy materials (carpets, fibrous glass ceiling panels or insulation, fabric-covered partitions). This reduces reverberation time and breaks direct paths of sound transmission. The partitions, where used, also provide some visual privacy and a feeling of occupant control (at least over the immediate work station area). However, they do substantially increase surface area and impede space air distribution.

Job Category and Type of Work

Lower status jobs were associated with higher complaint and symptom rates in the DTHS. Subordinate workers, such as clerical and drafting personnel, tend to spend more time at the work stations than their supervisors, who often move about the building or leave it to attend meetings. Lower status workers also tend to have less space and to be located near the interior of the building.

Outside air supply to the interior is usually less than at the perimeter. Some buildings deliver primarily or only recirculated air to the interior spaces. Thus, interior spaces may have stagnant or stale air. The combination of higher density, more activity, and less ventilation would result in higher concentrations of airborne contaminants. Perimeter offices would be more likely to have views out of windows, providing the worker with visual and psychological relief.

Type of Work. Certain jobs involve exposure to chemical and physical agents known to cause irritation, nervous system effects, and other health outcomes. Equipment and materials used in duplicating, printing, mailing, and clerical activities in general are all associated with various chemical and physical agents which may contaminate indoor air. It is also likely that individuals performing such work will be in subordinate positions and therefore at risk as discussed above.

Occupant Density and Number of Work Stations

It is not clear whether the DTHS found high occupant density associated with higher symptom report rates because of the density or the possible correlation of high density with job category and type of work, as described above. Lower status workers are likely to have less assigned personal work area, i.e., higher occupant density. At higher densities, the occupant-generated air contaminants (metabolic- and activity-based) will be more concentrated prior to dilution or removal by ventilation.

A higher number of work stations might be associated with increased anonymity, a lack of personal privacy and control, and a higher rate of generation of contaminants. All of these could affect occupant stress and comfort levels.

Woods (1988) has found a high proportion of buildings with high occupant densities that exceed the design capacities of the ventilation system. Occupants are added without modification to the ventilation system, resulting in inadequate air supply, interruptions of design airflows, and excessive loads on cooling and heating equipment.

LIMITATIONS OF INVESTIGATIONS

There are numerous limitations on investigations of problem buildings which suggest explanations for the frequent failure to identify etiologic agents or contributory building factors. Among these limitations are cost, timeliness, investigatory methods, building complexity, building dynamics, institutional contraints, and insufficient guidance to investigators.

Unsystematic and incomplete investigations result in inadequate diagnoses and unsuccessful remedial efforts. Yet complete, systematic investigations are rare. Expert investigators are usually selected by the occupants or building owner according to the owner's perceptions of the problem etiology. Expertise is usually confined to one or a limited number of fields, resulting in incomplete investigations and narrowly focused findings in many (Molhave 1987; Kreiss and Hodgson 1984).

The perseverance of the investigators and the availability of methods can determine whether chemical and biological agents can be eliminated as etiologies of building-associated outbreaks (Kreiss and Hodgson 1984). In many instances, ventilation system problems are identified early in the investigation. Modifications to the system equipment, operating schedule, or operational modes (airflow, temperature) will result in a significant reduction of complaints and symptoms, and the investigation will be terminated before problem causes are defined.

Timely investigations rarely occur due to institutional constraints. Frequently, when complaints or symptoms are initially reported, there is hesitation by management to give importance to them. It is often only when complaints become very numerous, when upper-level management personnel are affected, or when workers initiate organized or formal action that management commissions investigators.

Comprehensive and systematic investigations are expensive, difficult, and more time-consuming than building operators or users can normally tolerate. In many cases it is not considered necessary and it is not economically feasible to identify causes if remedial measures can effectively reduce complaints and symptoms, as in the case of ventilation system modifications. Thorough characterization of environmental factors, including detailed chemical and biological contaminant measurements, can be prohibitively expensive and is only undertaken in the most severe or persistent of cases.

Protocols to guide investigations of problem buildings have not been widely tested, validated, or promulgated by any standards development organization. While some generalized protocols have been prepared, significant differences among problem building cases require individualization of protocols and measurement methods (Levin 1987c; NAS 1985; NIOSH 1987; Sterling et al. 1987; Woods et al. 1988). A standard guide for investigation of problem buildings is currently being prepared by ASTM (Levin 1988).

Standardized sampling and analytical methods for indoor air are limited and those that exist are not uniformly applied or widely used. Efforts to address these shortcomings are under way by ASTM (Levin 1988). However, interbuilding variations limit standardization of investigations and comparability of results (Levin 1987c).

Comprehensive monitoring for airborne contaminants is extremely expensive and rarely definitive in problem building investigations. Monitoring for airborne VOC is expensive, and there is a lack of general agreement regarding appropriate monitoring methods. Characterization of total VOC is not as expensive as identification and quantification of specific compounds, although the methodological problems are significant.

Even where extensive environmental monitoring is conducted, interpretation of results is limited by the absence of guidelines and standards. Interpretation is often based upon standards or guidelines developed in and for different contexts, such as the industrial workplace and ambient air. Comparison of measurements to such guidelines or standards can lead to incorrect assumptions about the effect of the measured parameter on occupant health and comfort (Eisinger 1988).

Limitations Imposed by the Problem Context

Buildings are dynamic, responding to changes in the external environment, internal loads, and the building itself. Internal loads may be generated by user activities, building equipment, or occupants' appliances and equipment. Building loads vary as a result of normal operation of building equipment, principally lighting, ventilation, heating, and cooling. Malfunctions in building equipment, interventions by occupants, manipulation by building operators, and signals from building systems controls constantly effect changes in equipment operation resulting in load changes and load handling.

Environmental variations among locations within a single building can be enormous. Small distances between locations can involve large differences in some environmental variables including critical air quality factors. Significant variations in environmental factors occur hourly, daily, and seasonally. Therefore, monitoring of environmental factors, including but not limited to the sampling and analysis of indoor air, can produce misleading results unless an adequate number of representative samples is collected over extended or adequately representative time periods and locations. This may be an especially important factor in the failure of many large building or mutli-building studies where associations between reported symptoms and environmental variables are not found at statistically significant levels. Single area measurements or one measurement per floor or per ventilation system for many occupants will simply average precisely the variations that the study seeks to elucidate.

Designer, operator, and occupant perceptions frequently differ from each other and from actual building conditions. Investigator interviews with some but not all of these parties can result in biased assumptions or hypotheses followed by incomplete or poorly focused investigations.

DISCUSSION OF SICK BUILDING DIAGNOSES AND FINDINGS

By definition, an SBS diagnosis requires confirmation that an elevated complaint or symptom rate is associated with occupancy of a particular building, that no known etiology accounts for the symptoms, and that clinical evidence of building-related illness is absent.

SBS can be hypothesized but not diagnosed or defined based solely on clinical evidence unless sufficient numbers of occupants are examined in the case building and compared to occupants of a control building or to a valid baseline for symptom prevalence. Clinical identification of symptoms associated with occupancy of a particular building can result in an SBS hypothesis. Only an epidemiologic or clinical investigation can define the occurrence of SBS. Even where epidemiologic evidence supports an SBS hypothesis, BRI may be present in some or all of the occupants manifesting SBS symptoms.

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SBS may involve diverse reactions among building occupants. This may be a function of varying conditions within the building, varying individual responses to environmental factors, or both. Where this is the case, it will reduce statistical associations between reported symptoms and measured environmental conditions, and may lead to incorrect interpretations of even the most complete and careful investigations.

Therefore, as defined and discussed in this paper and elsewhere, SBS may not be a useful term in that it refers to symptom sets which are manifestations of various distinct illnesses or diseases. Aggregating these distinct medical conditions may be the greatest barrier to discovery of the causes of sick building syndrome.

Building Diagnostics

"Building diagnostics" is the name given to a set of practices used to assess the current performance and capability of a building and to predict its likely performance in the future (NAS 1985). While building diagnostics can be valuable at many stages in the life of a building, it may be most useful in investigations of problem buildings. Four elements are essential to building diagnostics, according to the NAS report; they are as follows:

(1) knowledge of what to measure,
(2) availability of appropriate instruments and other measurement tools,
(3) expertise in interpreting the measurements, and
(4) capability of predicting the future condition of the building based on that interpretation.

Several authors have proposed phased investigations of problem buildings or in other applications of building diagnostics (NAS 1985; Sterling et al. 1987; NIOSH 1987; Woods et al. 1988). Woods et al. (1988) have divided the phases as described below.

1. Consultation - scope of the investigation is defined and observations of the building and its systems are made (walkthrough survey). Few or no instrumented measurements are made.

Most advocates of phased diagnostic investigations of problem buildings urge extremely limited use of airborne monitoring during the initial phase. They assert that the majority of building problems can be solved or resolved without extensive monitoring. Furthermore, it is argued that monitoring is of limited effectiveness until it can be focused on hypothesized causal agents or factors.

2. Qualitative diagnostics - hypotheses are formulated through engineering analysis; system performance analysis is initiated with limited measurements (such as airflow and pressure differences). Medical evaluation identifies suspect pollutants and air or bulk samples will be collected for these substances.

3. Quantitative diagnostics - if further investigation is needed to test hypotheses, samples will be collected and analyzed and other environmental measurements will be made.

BUILDING ECOLOGY

Indoor air quality is beginning to receive recognition as an important indoor environmental factor as lighting, acoustics, privacy, security, thermal comfort, and aesthetics have received historically. An approach to understanding buildings and human health based on a systematic, comprehensive framework is badly needed. We recommended an ecological approach -- a methodology utilizing knowledge and analytical methods like those used by biological scientists in the study of living organisms in relationship to their environment. We have borrowed from the core of the definition of ecology to coin the term "building ecology," which we define as the study of buildings and their relationship to the natural and built environment around them and to humans who use or are otherwise affected by them (Levin 1981).

We suggest that the concepts of dynamic, interdependent flows used in studying ecosystems exemplify methods that can be adapted to the study of indoor air and human health. An example is a mass balance and mass flow analysis of a contaminant or of moisture into, through, and out of a building. Models for such mass balances and flows have shown that changes in one factor can shift the rate of the processes and the overall distribution of contaminants. Chaos theory in physics has shown that a small perturbation can initiate major deviations from a steady state or regular periodic behavior. Building environmental control systems designed to address these perturbations are themselves subject to changes resulting in small perturbations. There are time constants or lags for each of these changes as well. Thus, the system is an ever-shifting collection of interconnected entities -- in the case of the building, both living and inanimate; in the case of the ecosystems, each organism and its environment.

PREVENTING AND REMEDIATING PROBLEM BUILDINGS

Based on the causal factors identified in the Danish Town Hall Study and other investigations, we have identified some potential preventive or remedial measures to minimize SBS. These measures, which are listed in Table 9, have not been systematically evaluated, but there is considerable evidence to support their potential efficacy in reducing the occurrence of sick building syndrome.

CONCLUSION

Buildings are complex. Their effects on humans are extensive and are poorly understood. Attitudes toward and understandings of buildings' effects on occupant health and comfort are not generally shared.

Sick building syndrome is inadequately understood at this time, in part due to confusion in definitions and terminology, in part due to inadequate efforts to study comprehensively its occurrence and causes. Potential causes have been identified and discussed, but the definition of SBS may itself preclude elucidation of its causes due to the limitations on investigations. There are great difficulties inherent in conducting comprehensive investigations or studies which are sufficiently sensitive to detect relevant associations.

Causes of SBS require further investigation. An approach for conceptualizing and conducting problem building investigations has been outlined. We recommend that further efforts to develop and refine models for application in diagnosing problem buildings be developed. Field studies, laboratory studies, and modeling efforts need to be performed to further elucidate the causes and nature of sick building syndrome.

Potential control measures have been identified. Evaluation of the efficacy of potential control measures is warranted due to the large costs involved in their implementation and the risks attendant to their failure.

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Table 1. Common Features of Symptoms Reported in Cases of Sick Building Syndrome (WHO 1983).

Eye, Nose and Throat Irritation
Sensation of Dry Mucous Membranes and Skin
Erythema
Mental Fatigue
Headaches, High Frequency of Airway Infections and Cough
Hoarseness, Wheezing, Itching and Unspecific Hypersensitivity
Nausea, Dizziness

Table 2. Molhave's classification scheme for symptoms related to sick building syndrome and examples of each (Molhave 1987).

1. Sensoric irritation in eye, nose or throat
   - dryness
   - stinging, smarting, irritating sensation
   - hoarseness, changed voice
2. Skin irritation
   - reddening of skin
   - stinging, smarting, itching sensation
   - dry skin
3. Neurotoxic symptoms
   - mental fatigue
   - reduced memory
   - lethargy, drowsiness
   - reduced power of concentration
   - reduced memory
   - headache
   - dizziness, intoxication
   - nausea
   - tiredness
4. Unspecific hyperreactions
   - running nose and eye
   - asthma-like symptoms in non asthmatic persons
   - respiratory sounds
5. Odor and taste complaints
   - changed sensitivity
   - unpleasant odor or taste

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Table 3. WHO classification scheme for symptoms found in sick buildings (WHO 1986).

1. Sensory irritation of skin and upper airways, along with headache and abnormal taste
2. Odor
3. General symptoms such as fatigue, dizziness and nausea
4. Lower airway and gastrointestinal symptoms (*)

(*) Not generally found in sick building syndrome

Table 4. Proposed classification scheme for occupant condition in problem buildings (after Woods 1988).

1. Unresolved.
   Symptoms reported or complaint rates deemed unacceptable by owners, operators or occupants but do not meet standard statistical tests to confirm their association with occupancy of the building. This can occur where contamination or complaints are limited to a small area of a building or occur among the general building population but at rates similar to those found in buildings in general.

2. Building-associated illness.
   Complaint or symptom rates are elevated compared to control buildings or numbers derived from large population studies. Investigation confirms that the complaints are related to occupancy of the building.

   a) Sick building syndrome (SBS) or building sickness (BS). Symptoms are similar to those identified in Tables 1 and 2, but no specific cause of the complaints can be demonstrated by the investigators.

   b) Building-related illness (BRI).
   The disease entity is medically identified and verified. Contamination problems determined as directly associated with the disease(s) involve a wide range of agents and factors including infectious microorganisms, allergens, chemicals, moisture, temperature, noise, vibration and illumination.

   c) Unclassified building associated illness.

3. Undetected problem buildings

Table 5. Causal factors identifed in investigations of SBS (after Wallace 1988).

MULTIFACTORIAL
- Sex
- Hay fever
- Migraine
- Smoking
- Home-related illness
- Carbonless copy paper use
- Xeroxing > 25 sheets/d
- VDT use > 1 h/d
- Unsatisfied with job

PHYSICAL FACTORS
- Ventilation
- Ions
- Other physical causes

CHEMICAL FACTORS
- Formaldehyde
- Other volatile organic chemicals
- Semivolatile organic chemicals

BIOLOGICAL FACTORS
- Molds
- Bacteria
- Allergens

Table 6. Indoor climate measurements in 14 Danish town halls (Valbjorn and Skov 1987).

		Mean	Range Low	High
Mean external temperature	(24 hours)(°C)	2.4	-1.2	11.4
Average daily sunshine	(hours)	2.3	0	6.4
Air temperature	(°C)	22.7	20.5	24.1
Person-weighted air temperature	(°C)	23.0	22.0	24.4
Temperature rise during a work day	(°C)	2.5	1.0	8.0
Vertical temperature gradient	(°C/m)	0.9	0.9	2.0
Air velocity	(m/s)	0.15	<0.15	0.20
Relative humidity	(%)	32	25	40
CO_2	(%)	0.08	0.05	0.13
Formaldehyde	mg/m^3	0.04	0	0.08
Static Electricity: Observer	(kv)	1.4	0	4.8
Occupants max.	(kv)	1.7	0	4.0
Airborne dust	(mg/m^3)	0.201	0.086	0.382
Dust particles: >0.5 um	(l^{-1})	48×10^3	19×10^3	119×10^3
>2.0 um	(l^{-1})	25×10^2	8×10^2	116×10^2
Airborne microfungi	(col/m^3)	32	0	111
Airborne bacteria	(col/m^3)	574	120	2100
Airborne actinomycetes	(col/m^3)	4	0	15
Vacuum cleaned dust[a]	($g/12m^2$)	3.67	0.32	11.56
Vacuum cleaned dust[b]	($g/12m^2$)	6.14	0.66	17.04
Macromolecular content in the dust	(mg/g)	1.53	0	5.24
Macrofungi in the dust[a]	(col/30mg)	33	11	90
Macrofungi in the dust[b]	(col/30mg)	32	6	192
Bacteria in the dust[a]	(col/30 mg)	199	41	380
Bacteria in the dust[b]	(col/30 mg)	296	160	680
Man-made mineral fibers in air MMMF	(f/m^3)	5	0	60
Not MMMF (<3 um) in the air	($f \times 10^3/m^3$)[c]	33.2	18.5	59.1
Not MMMF (>3 um) in the air	($f \times 10^3/m^3$)[d]	3.1	0.7	5.0
Volatile Organic Compounds (charcoal)	(mg/m^3)	1.56	0.43	2.63
Volatile Organic Compounds (Tenax)	(mg/m^3)	0.5	0.1	1.2
A-weighted equivalent background noise, $L_{a,eq}$	(dB)	56.7	51.3	60.3
A-weighted equivalent background noise, L_{95}	(dB)	36.2	28.2	44.1
Reverberation time	(s)	0.41	0.28	1.05

[a] = In the office where all the measurements were performed
[b] = In an office with a considerable loading of clients during the day.
[c] = Mean readings in 6 buildings
[d] = Mean readings in 13 buildings, in one building measured 32 mg/m^3

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Table 7. Building and occupant factors (Valbjorn and Skov 1987) and their possible connection to the etiology of building sickness.

	ENVIRONMENTAL AND OCCUPANT EFFECTS	
FACTORS IDENTIFIED IN DANISH TOWN HALL STUDY	CO-FACTOR	HYPOTHESIZED FACTOR IN SBS ETIOLOGY
1. TEMPERATURE (elevated)	Microorganism proliferation	Bioaerosol increase
	Higher VOC emissions/air levels	VOC increase
	Reduced airflow	Contaminant increase
	Reduced ventilation	Increased contaminant levels
2. FLEECY MATERIAL	More VOC sources	Increased VOC levels
	More VOC adsorption surface area	Increased VOC levels
	More fiber sources	Increased airborne fibers
	Difficult housekeeping	Increased airborne dust
3. OPEN SHELVES	More adsorption sites	Increased VOC
	Fine particle deposition sites	Increased airborne particles
	More difficult housekeeping	Increased particles, VOC, bioaerosols
	More source surface area	
4. NEWER BUILDINGS <30 years	More fleecy surfaces	VOC, particles, aerosols
	Fewer private offices	Noise, crowding, contaminant levels
	Higher occupant density	Increased exposure to contaminants
	Less occupant control	Decreased resistance to illness
5. JOB CATEGORY	Less mobility during day	Increases exposure to contaminants
	Less control over time/work	Personal stress, reduced resistance
	Lower status	Higher exposure, less ventilation
	Less control over work area	Increased stress, reduced resistance
6. TYPE OF WORK	More exposure to toxins	More phsyiological stress
	More exposure to irritants	More physical and mental stress
	Stressful work posture	More physical stress
7. OCCUPANT DENSITY	Lack of privacy	Psychological stress
	Inadequate ventilation	Increased contaminant levels, exposure
	More local pollutant sources	Increased contaminant levels, exposure
8. #/WORK STATIONS	Anonymity, impersonal environment	Psychological stress
	Lack of privacy, control	Psychological stress
	More local pollutant sources	Increased exposure

Table 8. Identification of Possible Synergistic Risk Factors for SBS Based on Risk Factors in Danish Town Hall Study.

1. TEMPERATURE + FLEECY MATERIAL + OPEN SHELVES
   More organic sources, emissions
   More biological activity
2. JOB CATEGORY + TYPE OF WORK
   Low status = loss of control, mobility, job satisfaction
   Reduced proximity to windows - light, views, outside air
   Combined stressors of work posture, toxins, irritants
3. NUMBER OF WORK STATIONS + AGE OF BUILDING
   Higher density in open office plan, newer buildings
   More local sources
   Less privacy, control, space
4. SEX + JOB CATEGORY
   Females in subordinate (clerical) positions
   Type of work (see #2 above)

TABLE 9. POTENTIAL CONTROL MEASURES FOR MULTIFACTORIAL SBS

HARD SURFACES
Reduce surface area of materials exposed to interior,
Use building form, layout, and surface treatment for acoustic control.

LOWEST COMFORTABLE AIR TEMPERATURE
- Especially when building or furnishings are new or after floor and furniture polishing, waxing.

MINIMIZE OPEN STORAGE SHELVING
Use enclosed shelves where possible.
Locate shelves in separate space with exhaust ventilation, no recirculation.

LOW OCCUPANT DENSITY
Utilize total building space for roughly equal area per occupant. Avoid crowding. Confirm HVAC capacity for additional occupants.

MAXIMIZE OUTDOOR AIR SUPPLY
Extend hours and days of operation; increase percent outside air. Start-up earlier after days of vacancy.

INCREASE WORKER CONTROL, PRIVACY
Provide local and individual control over lighting, ventilation, heating, cooling, acoustic environment.

FLEXIBLE WORK HOURS
Allow individual schedule, where feasible.

MINIMIZE EXPOSURE IN STRESSFUL JOBS
Rotate jobs, mandatory rest (fresh air?) breaks.

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